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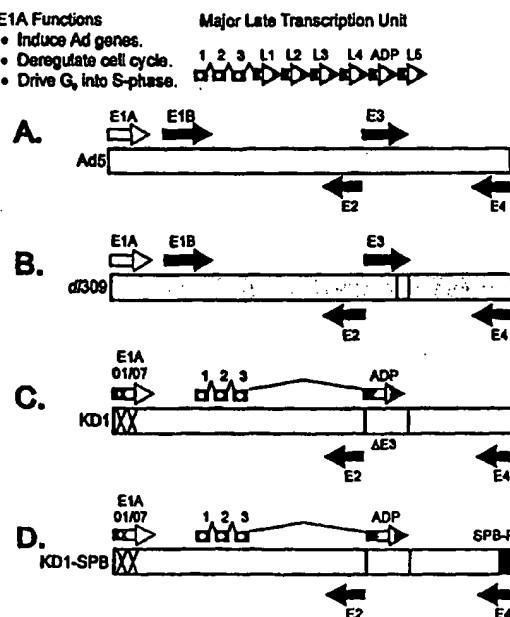
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(54) Title: REPLICATION-COMPETENT ANTI-CANCER VECTORS



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(57) Abstract: Novel vectors which are replication-competent in neoplastic cells and which overexpress an adenovirus death protein are disclosed. Some of the disclosed vectors are replication-restricted to neoplastic cells or to neoplastic alveolar type II cells. Compositions and methods for promoting the death of neoplastic cells using these replication-competent vectors are also disclosed.



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Replication-Competent Anti-Cancer Vectors

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5 Background of the Invention

(1) Field of the Invention

This invention relates generally to the treatment of cancer and more particularly to vectors which replicate in neoplastic cells and which overexpress an adenovirus death protein (ADP) and to the use of these vectors in treating human cancer.

10 (2) Description of the Related Art

Cancer is a leading cause of death in the United States and elsewhere. Depending on the type of cancer, it is typically treated with surgery, chemotherapy, and/or radiation. These treatments often fail: surgery may not remove all the cancer; some cancers are resistant to chemotherapy and radiation therapy; and chemotherapy-resistant tumors frequently develop.

15 New therapies are necessary, to be used alone or in combination with classical techniques.

One potential therapy under active investigation is treating tumors with recombinant viral vectors expressing anti-cancer therapeutic proteins. Adenovirus-based vectors contain several characteristics that make them conceptually appealing for use in treating cancer, as well as for therapy of genetic disorders. Adenoviruses (hereinafter used interchangeably with

"Ads") can easily be grown in culture to high titer stocks that are stable. They have a broad host range, replicating in most human cancer cell types. Their genome can be manipulated by site-directed mutation and insertion of foreign genes expressed from foreign promoters.

The adenovirion consists of a DNA-protein core within a protein capsid (reviewed by 5 Stewart et al., "Adenovirus structure by x-ray crystallography and electron microscopy." in: *The Molecular Repertoire of Adenoviruses*, Doerfler, W. et al., (ed.), Springer-Verlag, Heidelberg, Germany, p. 25-38). Virions bind to a specific cellular receptor, are endocytosed, and the genome is extruded from endosomes and transported to the nucleus. The genome is a linear duplex DNA of about 36 kbp, encoding about 36 genes (Fig. 1A). In the nucleus, the 10 10 "immediate early" E1A proteins are expressed initially, and these proteins induce expression of the "delayed early" proteins encoded by the E1B, E2, E3, and E4 transcription units (reviewed by Shenk, T. "Adenoviridae: the viruses and their replication" in: *Fields Virology*, Field, B.N. et al., Lippencott-Raven, Philadelphia, p. 2111-2148). E1A proteins also induce or repress cellular genes, resulting in stimulation of the cell cycle. About 23 early proteins 15 function to usurp the cell and initiate viral DNA replication. Viral DNA replicates at about 7 h post-infection (p.i.), then late genes are expressed from the "major late" transcription unit. Major late mRNAs are synthesized from the common "major late promoter" by alternative pre-mRNA processing. Each late mRNA contains a common "tripartite leader" at its 5'-terminus (exons 1, 2, and 3 in Fig. 1), which allows for efficient translation of Ad late 20 mRNAs. Cellular protein synthesis is shut off, and the cell becomes a factory for making viral proteins. Virions assemble in the nucleus at about 1 day p.i., and after 2-3 days the cell lyses and releases progeny virus. Cell lysis is mediated by the E3 11.6K protein, which has been renamed "adenovirus death protein" (ADP) (Tollefson et al., *J. Virol.* 70:2296-2306, 1996; Tollefson et al., *Virol.* 220:152-162, 1996). The term ADP as used herein in a generic 25 sense refers collectively to ADP's from adenoviruses such as, e.g. Ad type 1 (Ad1), Ad type 2 (Ad2), Ad type 5 (Ad5) or Ad type 6 (Ad6) all of which express homologous ADP's with a high degree of sequence similarity.

Human adenovirus type 5 (Ad5) is particularly useful for cancer gene therapy. It primarily causes asymptomatic or mild respiratory infections in young children, followed by 30 long term effective immunity. Fatalities are extremely rare except when the patient is immunocompromised (Horwitz, M. S., Adenoviruses, p. 2149-2171 *In* B. N. Fields, D. M. Knipe, and P. M. Howley (eds.), *Fields Virology*, Lippincott-Raven Publishers, Philadelphia, PA, 1996). Ad5 is very well understood, can be grown in culture to high titer stocks that are stable, and can replicate in most human cancer cell types (Shenk, T., *Adenoviridae: the 35 viruses and their replication*, p. 2111-2148. *In* B. N. Fields, D. M. Knipe, and P. M. Howley

(eds.), *Fields Virology*, Lippincott-Raven, Philadelphia, 1996). Its genome can be manipulated by site-directed mutagenesis and insertion of foreign sequences.

The Ad vectors being investigated for use in anti-cancer and gene therapy are based on recombinant Ad's that are either replication-defective or replication-competent. Typical 5 replication-defective Ad vectors lack the E1A and E1B genes (collectively known as E1) and contain in their place an expression cassette consisting of a promoter and pre-mRNA processing signals which drive expression of a foreign gene. The E1A proteins induce transcription of other Ad genes, and in nontransformed cells they deregulate the cell cycle, induce or repress a variety of cellular genes, and force cells from G₀ into S-phase 48 (White, 10 E., *Semin. Virol.* 8:505-513, 1998; Wold et al., pp. 200-232 *In* A.J. Cann (ed.), *DNA Virus Replication: Frontiers in Molecular Biology*, Oxford University Press, Oxford). The E1B proteins inhibit cellular apoptosis. *Id.* These vectors are unable to replicate because they lack the E1A genes required to induce Ad gene expression and DNA replication. In addition, the E3 genes are usually deleted because they are not essential for virus replication in cultured 15 cells.

A number of investigators have constructed replication-defective Ad vectors expressing anti-cancer therapeutic proteins. Usually, these vectors have been tested by direct injection of human tumors growing in mouse models. Most commonly, these vectors express the thymidine kinase gene from herpes simplex virus, and the mice are treated with 20 gancyclovir to kill cells transduced by the vector (see e.g., Felzmann et al., *Gene Ther.* 4:1322-1329, 1997). Another suicide gene therapy approach involves injecting tumors with a replication defective Ad vector expressing cytosine deaminase, followed by administration of 5-fluorocytosine (Topf et al., *Gene Ther.* 5:507-513, 1998). Investigators have also prepared and tested replication-defective Ad vectors expressing a cytokine-such as IL-2, IL-12, IL-6, 25 tumor necrosis factor (TNF), type I interferons, or the co-stimulatory molecule B7-1 in the anticipation that the Ad-expressed cytokine will stimulate an immune response, including cytotoxic T-lymphocytes (CTL), against the tumor (Felzmann et al., *supra*; Putzer et al., *Proc. Natl. Acad. Sci. USA* 94:10889-10894, 1997). Other vectors express tumor antigens (e.g. melanoma MART1), proteins that de-regulate the cell cycle and induce apoptosis (p53, pRB, 30 p21^{Kip1/WAF1}, p16^{CDKN2}, and even Ad E1A), and ribozymes. An Ad vector expressing FasL induces apoptosis and tumor regression of a mouse tumor (Arai et al., *Proc. Natl. Acad. Sci. USA* 94:13862-13867, 1997).

Despite these generally positive reports, it is recognized in the art that 35 replication-defective Ad vectors have several characteristics that make them suboptimal for use in therapy. For example, production of replication-defective vectors requires that they be grown on a complementing cell line that provides the E1A proteins in trans. Such cell lines

are fastidious, and generation of virus stocks is time-consuming and expensive. In addition, although many foreign proteins have been expressed from such vectors, the level of expression is low compared to Ad late proteins.

To address these problems, several groups have proposed using replication-competent Ad vectors for therapeutic use. Replication-competent vectors retain Ad genes essential for replication and thus do not require complementing cell lines to replicate. Replication-competent Ad vectors lyse cells as a natural part of the life cycle of the vector. Another advantage of replication-competent Ad vectors occurs when the vector is engineered to encode and express a foreign protein. Such vectors would be expected to greatly amplify synthesis of the encoded protein *in vivo* as the vector replicates. However, in order to prevent RC vectors from damaging normal tissues and causing disseminated viremia, it is important that they have some feature that limits their replication to cancer cells.

Wyeth Laboratories developed replication-competent Ad vectors for vaccination purposes, using vaccine strains of Ad serotypes 4, 7, and 5 (Lubeck et al., *AIDS Res. Hum. Retroviruses* 10:1443-1449, 1994). Foreign genes were inserted into the E3 region (with the E3 genes deleted) or into a site at the right end of the genome. Two foreign genes used were hepatitis B surface antigen and the HIV envelope protein. They obtained good expression in culture, and were able to raise antisera in animal models. Phase I human trials were ambiguous, and the project was mostly abandoned.

Onyx Pharmaceuticals recently reported on adenovirus-based anti-cancer vectors which are replication deficient in non-neoplastic cells but which exhibit a replication phenotype in neoplastic cells lacking functional p53 and/or retinoblastoma (pRB) tumor suppressor proteins (U.S. Patent No. 5,677,178; Heise et al., *Nature Med.* 6:639-645, 1997; Bischoff et al., *Science* 274:373-376, 1996). This phenotype is reportedly accomplished by using recombinant adenoviruses containing a mutation in the E1B region that make the encoded E1B-55K protein incapable of binding to p53 and/or a mutation(s) in the E1A region which make the encoded E1A protein (p289R or p243R) incapable of binding to pRB and/or the cellular 300 kD polypeptide and/or the 107 kD polypeptide. E1B-55K has at least two independent functions: it binds and inactivates the tumor suppressor protein p53, and it is required for efficient transport of Ad mRNA from the nucleus. Because these E1B and E1A viral proteins are involved in forcing cells into S-phase, which is required for replication of adenovirus DNA, and because the p53 and pRB proteins block cell cycle progression, the recombinant adenovirus vectors described by Onyx should replicate in cells defective in p53 and/or pRB, which is the case for many cancer cells, but not in cells with wild-type p53 and/or pRB. Onyx has reported that replication of an adenovirus lacking E1B-55K, which is named ONYX-015, was restricted to p53-minus cancer cell lines (Bischoff et al., *supra*), and

that ONYX-015 slowed the growth or caused regression of a p53-minus human tumor growing in nude mice (Heise et al., *supra*). Others have challenged the Onyx report claiming that replication of ONYX-015 is independent of p53 genotype and occurs efficiently in some primary cultured human cells (Harada and Berk, *J. Virol.* 73:5333-5344, 1999). It is now 5 known that ONYX-015 can replicate in cells with wild-type p53 (Goodrum et al., *J. Virol.* 72:9479-9490, 1998; Harada et al., *J. Virol.* 73:5333-5344, 1999; Hay et al., *Hum. Gene Ther.* 10:579-590, 1999; Rothmann et al., *J. Virol.* 72:9470-9478, 1998; Turnell et al., *J. Virol.* 73:2074-2083, 1999). ONYX-015 does not replicate as well as wild-type adenovirus because E1B-55K is not available to facilitate viral mRNA transport from the nucleus. Also, ONYX-10 015 expresses less ADP than wild-type virus (see Example 1 below).

As an extension of the ONYX-015 concept, a replication-competent adenovirus vector was designed that has the gene for E1B-55K replaced with the herpes simplex virus thymidine kinase gene (Wilder et al., *Gene Therapy* 6:57-62, 1999). The group that constructed this vector reported that the combination of the vector plus gancyclovir showed a 15 therapeutic effect on a human colon cancer in a nude mouse model (Wilder et al., *Cancer Res.* 59:410-413, 1999). However, this vector lacks the gene for ADP, and accordingly, the vector will lyse cells and spread from cell-to-cell less efficiently than an equivalent vector that expresses ADP. The gene for ADP is also lacking in another replication-competent adenovirus vector that has been described, in which a minimal enhancer/promoter of the 20 human prostate specific antigen was inserted into the adenovirus E1A enhancer/promoter (Rodriguez et al., *Cancer Res.* 57:2559-2563, 1997).

Another strategy for replication-competent vector improvement is to place replication under the control of tissue-specific promoters. One group replaced the basal E1A promoter with a modified promoter for α -fetoprotein (AFP) (Hallenbeck et al., *Hum. Gene Ther.* 10:1721-1733, 1999). AFP is expressed in the liver during development, but it is not 25 expressed in adults. However, it is expressed in 70-80% of patients with hepatocellular carcinoma. Growth of this vector was limited to AFP-expressing cells and the vector showed some suppression of xenotransplants. *Id.* A series of RC vectors has also been developed that have expression of the E1A and E1B genes dependent on the prostate tumor-specific 30 prostate specific antigen (PSA) and kallikrein promoters/enhancers (Rodriguez et al., *Cancer Res.* 60:1196, 1997; Yu et al., *Cancer Res.* 59:4200-4203, 2000; Yu et al., *Cancer Res.* 59:1498-1504, 1999).

Thus, there is a continuing need for vectors that replicate and spread efficiently in tumors but that can be modified such that they replicate poorly or not at all in normal tissue.

35 Summary of the Invention

Briefly, therefore, the present invention is directed to novel vectors which are replication competent in neoplastic cells and which overexpress an adenovirus death protein (ADP). The work reported herein demonstrates the discovery that overexpression of ADP by a recombinant adenovirus allows the construction of a replication-competent adenovirus that kills neoplastic cells and spreads from cell-to-cell at a rate similar to or faster than that exhibited by adenoviruses expressing wild-type levels of ADP, even when the recombinant adenovirus contains a mutation that would otherwise reduce its replication rate in non-neoplastic cells. This discovery was unexpected because it could not have been predicted from what was known about adenovirus biology that Ad vectors overexpressing ADP remain viable and that the infected cells are not killed by the higher amounts of ADP before the Ad vector produces new virus particles that can spread to other tumor cells. Indeed, naturally-occurring adenoviruses express ADP in low amounts from the E3 promoter at early stages of infection, and begin to make ADP in large amounts only at 24-30 h p.i., once virions have been assembled in the cell nucleus. It is believed that other non-adenoviral vectors can be used to deliver ADP's cell-killing activity to neoplastic cells, including other viral vectors and plasmid expression vectors.

Thus, in one preferred embodiment, the ADP-expressing vector comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α (also known as 10.4K); RID β (also known as 14.5K) and 14.7K. Because these E3 proteins inhibit immune-mediated inflammation and/or apoptosis of Ad-infected cells, it is believed that a recombinant adenovirus lacking one or more of these E3 proteins will stimulate infiltration of inflammatory and immune cells into a tumor treated with the adenovirus and that this host immune response will aid in destruction of the tumor as well as tumors that have metastasized. The ADP expressed by preferred embodiments comprises a naturally-occurring amino acid sequence from a human adenovirus of subgroup C, namely Ad1, Ad2, Ad5 and Ad6.

In another embodiment, replication of the vector is restricted to neoplastic cells. Such replication-restricted vectors are useful in treating cancer patients in which it is desirable to eliminate or reduce damage to normal cells and tissues that might be caused by the vector, particularly viral vectors that kill the host cell as part of their life cycle. In preferred embodiments, a recombinant adenovirus has a replication-restricted phenotype because the recombinant adenovirus is incapable of expressing an E1A viral protein which binds the pRB and the p300/CBP proteins or because the E4 promoter has been substituted with a promoter that is activated only in neoplastic cells and/or cells of a specific tissue.

In yet another embodiment, the invention provides a vector which overexpresses ADP and whose replication is under the control of a tissue specific promoter, tumor specific

promoter or an inducible promoter. In preferred embodiments, the vector comprises a recombinant adenovirus in which the tissue specific promoter or inducible promoter is substituted for the E4 promoter. Such vectors are useful for restricting replication of the vector and its ADP-mediated cell killing to cells of a particular type or to cells exposed to an exogenous agent that activates the promoter. A preferred tissue-specific or inducible vector also expresses a phenotype that restricts its replication to neoplastic cells.

In yet another embodiment, the invention provides a vector which overexpresses ADP but which is not restricted to tumors by a specific genetic modification. Such a vector is more destructive to neoplastic cells than even the naturally occurring Ad's of subgroup C. In preferred embodiments, this vector could be used for patients with terminal cancer not treatable by another method, and who have pre-existing neutralizing antibodies to Ad or to which neutralizing antibodies can be administered.

In still another embodiment, the invention provides a composition comprising a first recombinant virus which is replication competent in a neoplastic cell and overexpresses the adenovirus death protein. In one embodiment, the recombinant virus is contained within a delivery vehicle comprising a targeting moiety that limits delivery of the virus to cells of a certain type. With this embodiment, the replication-competent vector can be of any ADP-overexpressing configuration described herein. In some embodiments, the composition also comprises a second recombinant virus which is replication-defective and which expresses an anti-cancer gene product. In some embodiments, the replication-defective vector may be engineered to overexpress ADP when replication of this vector is complemented by a replication-competent vector. The recombinant virus complements spread of the replication-defective virus, as well as its encoded anti-cancer product, throughout a tumor. In preferred embodiments, the first recombinant virus is a recombinant adenovirus whose replication is restricted to neoplastic cells and/or which lacks expression of one or more of the E3 gp19K; RID α ; RID β ; and 14.7K proteins.

In additional embodiments, the invention provides replication-competent vectors that overexpresses an ADP and also expresses an anti-cancer product. As with previous embodiments, the vector can be of any ADP-overexpressing configuration provided herein. Preferably, replication of the virus is engineered to (a) be restricted to neoplastic cells, e.g., by replacing the E4 promoter with a tissue specific or tumor specific promoter and/or (b) lack expression of one or more of the E3 gp19K; RID α ; RID β ; and 14.7K proteins. In some embodiments, the anti-cancer product is inserted into the E3 region.

The ADP-expressing vectors and compositions of the invention are useful in a method for promoting death of a neoplastic cell. The method comprises contacting the neoplastic cell with a vector which is replication-competent in the neoplastic cell and which

overexpresses ADP. Where the neoplastic cell comprises a tumor in a patient, the vector is administered directly to the tumor or, in other embodiments, the vector is administered to the patient systemically or in a delivery vehicle containing a targeting moiety that directs delivery of the vector to the tumor. In embodiments where the vector is a recombinant virus, the 5 method can also comprise passively immunizing the patient against the virus.

In yet another embodiment of the invention, the vector may be used in combination with radiation therapy. The radiation therapy can be any form of radiation therapy used in the art such as for example, external beam radiation such as x-ray treatment, radiation delivered by insertion of radioactive materials within the body near or at the tumor site such as 10 treatment with gamma ray emitting radionuclides, particle beam therapy which utilizes neutrons or charged particles and the like. In addition, this embodiment encompasses the use of more than one of the vectors of the present invention in a cocktail in combination with radiation therapy.

Another embodiment of the invention involves the use of the recombinant vector in 15 combination with chemotherapy as has been disclosed for other adenovirus vectors (U.S. Patent No. 5,846,945). Chemotherapeutic agents are known in the art and include antimetabolites including pyrimidine-analogue and purine-analogue antimetabolites, plant alkaloids, antitumor antibiotics, alkylating agents and the like. The use of more than one of the vectors of the present invention with a chemotherapeutic agent or agents is also 20 contemplated within this embodiment.

Among the several advantages found to be achieved by the present invention, therefore, may be noted the provision of replication-competent vectors, particularly viruses, which rapidly kill cancer cells and spread from cell-to-cell in a tumor; the provision of such vectors whose replication can be induced or which is restricted to tumors and/or to cells of a 25 certain tissue type; and the provision of compositions and methods for anti-cancer therapy which cause little to no side effects in normal tissues.

Brief Description of the Drawings

Figure 1 is a schematic of gene expression in Ad5 (Fig. 1A) and KD3, a preferred embodiment of the invention (Fig. 1B), in which the respective genomes are represented by 30 the stippled bars and transcription units represented by arrows above and below the bars, with the E3 proteins listed above the arrows for the E3 transcription unit, and the L1 to L5 families of late mRNA's indicated.

Figure 2 illustrates the overexpression of ADP by KD1, KD3, GZ1, and GZ3 showing an immunoblot of proteins isolated from human A549 cells infected with the 35 indicated viruses and probed with an anti-ADP antibody, with ADP indicating differently glycosylated and proteolytically processed forms of ADP.

Figure 3 illustrates that the E1A *d11101/1107* mutation referred to in the figure and hereinafter as *d101/07*, retards expression of late proteins, showing an immunoblot of E1A proteins and late proteins in A549 cells infected with the indicated viruses in the absence (Figs. 3A and 3B) or presence (Figs. 3C and 3D) of *d1327*, which has a wild-type E1A region and has a deletion of all E3 genes but the gene encoding the 12.5K protein (Figs. 3C and 3D). An antiserum specific to the E1A proteins was used for Fig. 3A and 3C. An antiserum raised against Ad5 virions was used for Figs. 3B and 3D.

Figure 4 illustrates that KD1 and KD3 kill cells more efficiently than control viruses that express less or no ADP, showing a graph of the percent of A549 cells infected with the indicated viruses that were viable at the indicated days p.i. as determined by trypan blue exclusion.

Figure 5 is a cell spread assay illustrating that overexpression of ADP enhances spread of virus from cell to cell, showing monolayers infected with the indicated viruses at the indicated PFU/cell which were treated at 7 days p.i. with crystal violet, which stains live cells but not dead cells.

Figure 6 illustrates that KD1 and KD3 replicate well in growing cells but not in growth-arrested cells showing the virus titer extracted from growing or growth arrested HEL-229 cells at various times following infection with 100 PFU/ml of the following viruses: *d1309* (Fig. 6A), *d101/07* (fig. 6B), KD1 (Fig. 6C) and KD3 (Fig 6D).

Figure 7 illustrates that KD1 and KD3 are defective in killing primary human bronchial epithelial cells showing these cell monolayers infected at 30% confluence with 10 PFU/ml of the indicated viruses and stained at 5 days p.i. with neutral red.

Figure 8 illustrates that KD1 and KD3 reduce the growth rate of human A549 cell tumors growing in nude mice, showing in Fig. 8A a graph of average-fold increase in tumor size plotted against the number of weeks following infection of the tumor with buffer or with 5×10^7 PFU at weekly intervals of or the indicated viruses, and showing in Fig. 8B a similar graph of tumors injected once with 5×10^8 PFU of KD3 or GZ3.

Figure 9 illustrates that KD1 and KD3 reduce the growth rate of human Hep3B cell tumors growing in nude mice, showing a graph of average-fold increase in tumor size plotted against the number of weeks following injection of the tumor with buffer or with 5×10^7 PFU of *d1309*, KD1 or KD3 at twice weekly intervals of the indicated viruses.

Figure 10 illustrates that KD1 and KD3 complement the replication and spread of Ad- β -gal, a replication-defective vector that expresses β -galactosidase, using an infectious center assay showing in Fig. 10A a picture of A549 cell monolayers seeded with A549 cells infected with Ad- β -gal alone or with the indicated viruses, with Figs 10B and 10C showing close-up views of two of the monolayers of Fig. 10A.

Figure 11 is a bar graph illustrating that KD1 and KD3 increase the expression of luciferase in human Hep3B cell tumors growing in nude mice, using an assay in which tumors were injected with the indicated combinations of viruses, then were extracted 2 weeks p.i. and assayed for luciferase activity. The numbers in parentheses indicated the fold increase in luciferase activity compared to that of the Adluc vector plus buffer.

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Figure 12 is a graph showing the results of a standard plaque development assay for KD1 and KD1-SPB on A549 cells engineered to express the TTF1 transcription factor (A549/TTF1) and the parental 549 cells, in which data are plotted as the number of plaques observed on a particular day in the assay divided by the final number of plaques observed for 10 that virus multiplied by 100.

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Figure 13 is a cell spread assay for KD1 and KD1-SPB on H441 cells and Hep3B cells, where cells were infected with the indicated amounts of KD1 or KD1-SPB and H441 cells and Hep3B cells were strained with crystal violet at 5 days p.i. and 8 days p.i., respectively.

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Figure 14 is a graph showing the results of a standard plaque development assay for *dl309* and two preferred embodiments of the invention, GZ1 and GZ3, in which data are plotted as the number of plaques observed on a particular day in the assay divided by the final number of plaques observed for that virus multiplied by 100.

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Figure 15 is a cell spread assay illustrating that the combination of KD1, KD3, GZ1, or GZ3 with x-ray radiation is more effective in destroying A549 cell monolayers than is virus vector alone or radiation alone, wherein cells were infected with the indicated amounts of the indicated viruses, radiated with 600 centigreys (cGy) of x-radiation (bottom panel), or mock radiated (top panel), then stained with crystal violet at 6 days p.i.

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Figure 16 is a graph of a cell spread assay illustrating that 10^3 PFU of KD1, KD3, GZ1, or GZ3 used in combination with 150, 300, or 600 centigreys of radiation is more effective in destroying A549 cell monolayers than virus vector alone or radiation alone. Cell viability is based on the amount of crystal violet extracted from the culture wells, using the mock-infected non-radiated well as 100% viability.

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Figure 17 illustrates that the combination of KD3 or GZ3 plus x-ray radiation is more effective in reducing the growth of A549 cell tumors growing in nude mice than KD3 alone or GZ3 alone.

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Figure 18 illustrates a structure-function analysis of ADP, showing in Fig. 18A the amino acid sequence of the adenovirus death protein encoded by Ad2, with the various putative domains and glycosylation sites labeled and showing in Fig. 18B a schematic of the ADP gene in *rec700* and in the indicated deletion mutants, with the right column

summarizing the death promoting phenotype of the various mutants as a percentage of the wild-type phenotype.

Figures 19A and 19B illustrate a cell viability assay of the indicated ADP mutants showing a graph of viability as determined by trypan blue exclusion plotted against hours (Fig. 19A) or days (Fig. 19B) postinfection.

Figure 20 depicts the amino acid sequence, shown in single letter code, for the ADP proteins of Ad1, Ad2, Ad5, and Ad6 (SEQ ID NOS:5-8), for the Ad2 ADP mutants *dl716*, *dl715*, *dl714*, and *dl737* (SEQ ID NOS:9-12), and for the putative luminal domain (SEQ ID NO:17), the transmembrane domain (SEQ ID NO:18), the cytosolic basic-proline domain (SEQ ID NO:19), and the remainder of the cystosolic domain (SEQ ID NO:20) of the ADP protein of Ad2.

Figure 21 presents the complete nucleotide sequence of the genome of Ad5.

Figure 22 presents the complete nucleotide sequence of the genome of KD1 (SEQ ID NO:1).

Figure 23 presents the complete nucleotide sequence of the genome of KD3 (SEQ ID NO:2).

Figure 24 is a schematic of the following vectors: A. Ad5. The stippled bar indicates the DNA genome of 36 kbp. The open arrow indicates the immediate early E1A transcription unit, and the black arrows are the delayed early E1B, E2, E3, and E4 transcription units. The hatched arrows indicate the five families of major late mRNAs, and also the ADP mRNA, which is synthesized as part of the major late transcription unit. Each major late mRNA has a tripartite leader (leaders 1, 2, and 3) spliced to its 5' terminus. B. *dl309*. *dl309* is identical to Ad5 except it has the E3-RID and E3-14.7K genes deleted. *dl309* expresses ADP at levels similar to Ad5. C. KD1. KD1 has two small deletions (indicated by "X" marks) in the E1A gene that abolish binding of the E1A proteins to pRB or p300/CBP. It lacks all E3 genes except *adp*. ADP is expressed earlier in infection and in greater abundance than is ADP from Ad5 or *dl309* Doronin et al., *J. Virol.* 74:6147-6155. D. KD1-SPB. KD1-SPB is identical to KD1, except it has the E4 promoter replaced by the promoter for Surfactant Protein B (SPB-P).

Figure 25 presents graphs illustrating that KD1-SPB grows as well as KD1 in H441 lung carcinoma cells but much more poorly than KD1 in Hep 3B hepatoma cells. CsCl-banded stocks of KD1-SPB and KD1 were titered using standard methods (Tollefson et al., p. 1-9 In W.S.M. Wold (ed.), *Adenovirus Methods and Protocols*. Humana Press, Inc., Totowa, NJ, 1998) on 293-E4 or 293 cells (A), or on A549 cells (B). The data are plotted as the number of plaques seen on any day of the plaque assay as a percentage of the number of plaques seen on the final day of the assay (Tollefson et al., *Virology* 220:152-162, 1996).

Figure 26 presents micrographs illustrating that KD1-SPB induces CPE in H441 cells but not Hep 3B cells. H441 and Hep 3B monolayers were mock-infected or infected with 10 PFU/cell of KD1 or KD1-SPB, then photographed under phase contrast at 4 or 7 days p.i.

Figure 27 depicts Southern hybridizations and a graph illustrating that KD1-SPB DNA is synthesized efficiently in H441 but not Hep 3B cells. H441 or Hep 3B cells were infected with 10 PFU/cell of KD1 or KD1-SPB. Total genomic DNA was isolated at 0, 5, 24, 48, 72, and 96 h p.i., digested with HindIII, resolved by agarose gel electrophoresis, blotted, and hybridized with ³²P-labeled Ad DNA. A. Autoradiogram. B. PhosphorImager quantitation of the DNA bands in Panel A.

Figure 28 presents graphs depicting single step growth curves showing that KD1-SPB grows well in H441 but not Hep 3B cells. Cells were infected with 10 PFU/cell of KD1 or KD1-SPB. Vectors were extracted at the indicated days p.i. and titers determined by plaque assay.

Figure 29 depicts immunoblots showing that KD1-SPB expresses E4ORF3 and ADP in H441 but not Hep 3B cells. Cells were infected with 10 PFU/cell of KD1 or KD1-SPB. At 24 h p.i., protein extracts were analyzed for E1A, E4ORF3, and ADP using specific antisera. The E1A proteins appear as multiple bands. ADP appears as two bands; the upper band is glycosylated and the lower band is a proteolytically cleaved species (Scaria et al., *Virology* 191:743-753, 1992; Tollefson et al., *J. Virol.* 66:3633-3642).

Figure 30 depicts immunofluorescence micrographs showing that KD1-SPB expresses E4ORF3 in H441 but not Hep 3B cells. Cells growing on coverslips were infected with 20 PFU/cell of KD1, KD1-SPB, or dl309 (wild-type). At 48 h (Panel A) or 6 days (Panel B), cells were fixed and stained with a rabbit polyclonal antipeptide antiserum against E4ORF3. Photographs were taken using a 100X Planapo lens. Each panel shows about 8 nuclei. This figure is part of the same experiment shown in Figure 31.

Figure 31 depicts immunofluorescence micrographs showing that KD1-SPB does not express E2-DBP or fiber efficiently in Hep 3B cells. Hep 3B cells were infected with 20 PFU/cell of KD1-SPB or KD1. At 48 h (A) or 6 days (B) p.i., cells were fixed and double-stained using a rabbit polyclonal antiserum against DBP and a mouse monoclonal antibody against fiber. The same fields are shown for DBP and fiber. This figure is part of the same experiment shown in Figure 30.

Figure 32 presents graphs illustrating that KD1-SPB lyses H441 but not Hep 3B as efficiently as KD1. H441 or Hep 3B cells were mock-infected or infected with 20 PFU/cell of KD1 or KD1-SPB. Cell lysis was determined by release of lactate dehydrogenase from the cells into the medium.

Figure 33 presents graphs illustrating that KD1-SPB suppresses growth of H441 tumors in nude mice equally as well as KD1. Tumor cells were injected into flanks of nude mice and allowed to grow to about 100 μ l (H441) or 150 μ l (Hep 3B) volumes. Tumors ($n = 10$) were injected with DMEM (mock) or with 5×10^7 PFU of KD1 or KD1-SPB. Injections of the viruses were repeated twice weekly for 3 weeks to a total dose of 3.0×10^8 PFU per tumor. Tumors were measured and the mean fold-increase in tumor size was calculated.

5 Description of the Preferred Embodiments

In accordance with the present invention, it has been discovered that overexpression of ADP by a recombinant adenovirus results in faster lysis of cells and spread of the virus throughout a cell monolayer than viruses expressing wild-type levels of ADP. It has also been discovered that this function for ADP is manifest in an adenovirus that contains E1A mutations that restrict adenoviral replication to neoplastic cells. Thus, vectors which are both replication competent in neoplastic cells and which overexpress ADP should be useful in anti-cancer therapy.

15 In the context of this disclosure, the following terms will be defined as follows unless otherwise indicated:

"Naturally-occurring" as applied to an object such as a polynucleotide, polypeptide, or virus means that the object can be isolated from a source in nature and has not been intentionally modified by a human.

20 "Neoplastic cell" means a cell which exhibits an aberrant growth phenotype characterized by a significant loss of control of cell proliferation and includes actively replicating cells as well as cells in a temporary non-replicative resting state (G_1 or G_2). A neoplastic cell may have a well-differentiated phenotype or a poorly-differentiated phenotype and may comprise a benign neoplasm or a malignant neoplasm.

25 "Recombinant virus" means any viral genome or virion that is different than a wild-type virus due to a deletion, insertion, or substitution of one or more nucleotides in the wild-type viral genome. The recombinant virus can have changes in the number of amino acid sequences encoded and expressed or in the amount or activity of proteins expressed by the virus. In particular, the term includes recombinant viruses generated by the intervention of a human.

30 "Replication-competent" as applied to a vector means that the vector is capable of replicating in normal and/or neoplastic cells. As applied to a recombinant virus, "replication-competent" means that the virus exhibits the following phenotypic characteristics in normal and/or neoplastic cells: cell infection; replication of the viral genome; and production and release of new virus particles; although one or more of these characteristics need not occur at the same rate as they occur in the same cell type infected by a wild-type virus, and may occur

at a faster or slower rate. Where the recombinant virus is derived from a virus such as adenovirus that lyses the cell as part of its life cycle, it is preferred that at least 5 to 25% of the cells in a cell culture monolayer are dead 5 days after infection. Preferably, a replication-competent virus infects and lyses at least 25 to 50%, more preferably at least 75%, and most preferably at least 90% of the cells of the monolayer by 5 days post infection (p.i.).

5 "Replication-defective" as applied to a recombinant virus means the virus is incapable of, or is greatly compromised in, replicating its genome in any cell type in the absence of a complementing replication-competent virus. Exceptions to this are cell lines such as 293 cells that have been engineered to express adenovirus E1A and E1B proteins.

10 "Replication-restricted" as applied to a vector of the invention means the vector replicates better in a dividing cell, i.e. either a neoplastic cell or a non-neoplastic, dividing cell, than in a cell of the same type that is not neoplastic and/or not dividing, which is also referenced herein as a normal, non-dividing cell. Preferably, a replication-restricted virus kills at least 10% more neoplastic cells than normal, non-dividing cells in cell culture
15 monolayers of the same size, as measured by the number of cells showing cytopathic effects (CPE) at 5 days p.i. More preferably, between 25% and 50%, and even more preferably, between 50% and 75% more neoplastic than normal cells are killed by a replication-restricted virus. Most preferably, a replication-restricted adenovirus kills between 75% and 100% more neoplastic than normal cells in equal sized monolayers by 5 days p.i.

20 In one embodiment the invention provides a vector that is replication-competent in neoplastic cells and which overexpresses an ADP. Vectors useful in the invention include but are not limited to plasmid-expression vectors, bacterial vectors such as *Salmonella* species that are able to invade and survive in a number of different cell types, vectors derived from DNA viruses such as human and non-human adenoviruses, adenovirus associated viruses
25 (AAVs), poxviruses, herpesviruses, and vectors derived from RNA viruses such as retroviruses and alphaviruses. Preferred vectors include recombinant viruses engineered to overexpress an ADP. Recombinant adenoviruses are particularly preferred for use as the vector, especially vectors derived from Ad1, Ad2, Ad5 or Ad6.

30 Vectors according to the invention overexpress ADP. As applied to recombinant Ad and AAV vectors, the term "overexpresses ADP" means that more ADP molecules are made per viral genome present in a dividing cell infected by the vector than expressed by any previously known recombinant adenoviral vector or AAV in a dividing cell of the same type. As applied to other, non-adenoviral vectors, "overexpresses ADP" means that the virus expresses sufficient ADP to lyse a cell containing the vector.

35 Vectors overexpressing ADP can be prepared using routine methodology. See, e.g., *A Laboratory Cloning Manual*, 2nd Ed., vol. 3, Sambrook et al., eds., Cold Spring Harbor

Laboratory Press, 1989. For example, a polynucleotide encoding the ADP can be cloned into a plasmid expression vector known to efficiently express heterologous proteins in mammalian cells. The polynucleotide should also include appropriate termination and polyadenylation signals. Enhancer elements may also be added to the plasmid to increase the amount of ADP expression. Viral vectors overexpressing ADP can be prepared using similar materials and techniques.

Where the virus is a recombinant adenovirus, overexpression of ADP can be achieved in a multitude of ways. In general, any type of deletion in the E3 region that removes a splice site for any of the E3 mRNAs will lead to overexpression of the mRNA for ADP, inasmuch as more of the E3 pre-mRNA molecules will be processed into the mRNA for ADP. This is exemplified in the KD1, KD3, GZ1 and GZ3 vectors (SEQ ID NOS:1-4) whose construction is described below. Other means of achieving overexpression of ADP in Ad vectors include, but are not limited to: insertion of pre-mRNA splicing and cleavage/polyadenylation signals at sites flanking the gene for ADP; expression of ADP from another promoter, e.g. the human cytomegalovirus promoter, inserted into a variety of sites in the Ad genome; and insertion of the gene for ADP behind the gene for another Ad mRNA, together with a sequence on the 5' side of the ADP sequence that allows for internal initiation of translation of ADP, e.g. the Ad tripartite leader or a viral internal ribosome initiation sequence.

The ADP expressed by a vector according to the invention is any polypeptide comprising a naturally-occurring full-length ADP amino acid sequence or variant thereof that confers upon a vector expressing the ADP the ability to lyse a cell containing the vector such that replicated copies of the vector are released from the infected cell. A preferred full-length ADP comprises the ADP amino acid sequence encoded by Ad1, Ad2, Ad5 or Ad6. These naturally-occurring ADP sequences are set forth in SEQ ID NOS:5-8, respectively. ADP variants include fragments and deletion mutants of naturally-occurring adenovirus death proteins, as well as full-length molecules, fragments and deletion mutants containing conservative amino acid substitutions, provided that such variants retain the ability, when expressed by a vector inside a cell, to lyse the cell.

Conservative amino acid substitutions refer to the interchangeability of residues having similar side chains. Conservatively substituted amino acids can be grouped according to the chemical properties of their side chains. For example, one grouping of amino acids includes those amino acids having neutral and hydrophobic side chains (A, V, L, I, P, W, F, and M); another grouping is those amino acids having neutral and polar side chains (G, S, T, Y, C, N, and Q); another grouping is those amino acids having basic side chains (K, R, and H); another grouping is those amino acids having acidic side chains (D and E); another grouping is those amino acids having aliphatic side chains (G, A, V, L, and I); another

grouping is those amino acids having aliphatic-hydroxyl side chains (S and T); another grouping is those amino acids having amine-containing side chains (N, Q, K, R, and H); another grouping is those amino acids having aromatic side chains (F, Y, and W); and another grouping is those amino acids having sulfur-containing side chains (C and M). Preferred 5 conservative amino acid substitutions groups are: R-K; E-D, Y-F, L-M; V-I, and Q-H.

As used herein, an ADP variant can also include modifications of a naturally- occurring ADP in which one or more amino acids have been inserted, deleted or replaced with a different amino acid or a modified or unusual amino acid, as well as modifications such as glycosylation or phosphorylation of one or more amino acids so long as the ADP variant 10 containing the modified sequence retains cell lysing activity.

As described below, the inventors herein performed a structure-function analysis of ADP that defined specific domains in ADP required to promote cell death. Using this information, when combined with known recombinant DNA and cloning methodology, it is believed the skilled artisan can readily construct ADP variants of a naturally-occurring 15 adenovirus death protein and test them for cell lysing activity. A preferred ADP deletion mutant comprises an ADP amino acid sequence from any of the deletion mutants *dl716*, *dl715*, *dl714* and *dl737*, whose ADP sequences are set forth in SEQ ID NOS:9-12, respectively).

Where the vector is derived from a virus, it is preferred that the virus lack expression 20 of one or more viral proteins involved in avoiding host anti-viral defenses such as immune-mediated inflammation and/or apoptosis of infected cells. For example, adenovirus contains a cassette of genes that prevents killing of Ad-infected cells by the immune system (Wold et al., *Semin. Virol.*, 1998 (8:515-523, 1998). The E3-14.7K protein and the E3 RID (Receptor Internalization and Degradation) protein, which is a complex consisting of RID α and RID β , inhibit apoptosis of Ad-infected cells induced by tumor necrosis factor (TNF) and the Fas 25 ligand which are expressed on, or secreted by, activated macrophages, natural killer (NK) cells, and cytotoxic lymphocytes (CTLs) (Tollefson et al., *Nature* 392:727-730, 1998). The E3-gp19K protein inhibits CTL-killing of infected cells by blocking transport of MHC class I antigens to the cell surface (Wold et al., *supra*). Thus, it is believed that infection of tumor 30 cells by such viral vectors will stimulate infiltration of inflammatory cells and lymphocytes into the tumor, and will not prevent infected tumor cells from apoptosis induced by cytolytic cells of the immune system, or against apoptosis inducing cytokines. For example, it is known that when mice are infected with Ad mutants lacking the E3 gp19K, RID and 14.7K proteins there is a dramatic increase (as compared to E3-positive Ad) in infiltration of 35 inflammatory cells and lymphocytes into the infected tissue (Sparer et al., *J. Virol.* 70:2431-2439, 1996). A similar infiltration of tumors infected by an ADP-expressing viral vector of

the invention would be expected to further promote destruction of the tumor by adding an immune system attack to the ADP-mediated killing activity. For example, it is believed that the viral infection will stimulate formation of tumor-specific CTL's that can kill neoplastic cells not only in the tumor but also ones that have metastasized. In addition, it is also 5 expected that vector-specific CTL's will be generated which could attack vector-infected cells if the vector spreads away from the tumor into normal cells. Because viral vectors overexpressing ADP will spread rapidly through the tumor, it is believed these immune mechanisms will have little effect on spread of the vector.

Where the vector is a recombinant adenovirus, it is preferred that the adenovirus lack 10 expression of each of the E3 gp19K, RID, and 14.7K proteins. By "lack expression" and "lacking expression" of a protein(s), it is meant that the viral genome contains one or more mutations that inactivates expression of a functional protein, i.e., one having all the functions of the wild-type protein. The inactivating mutation includes but is not limited to substitution or deletion of one or more nucleotides in the encoding gene(s) that prevents expression of 15 functional transcripts or that results in transcripts encoding nonfunctional translation products. A particularly preferred way to inactivate expression of the Ad E3 gp19K, RID, and 14.7K proteins is by deleting the E3 region containing the genes encoding these proteins. Preferably, one or both of the E3 genes encoding the E3 6.7K and 12.5K proteins are also deleted because, as discussed in the Examples below, it is believed that deletion of most or all 20 of the E3 genes other than the ADP gene facilitates overexpression of ADP mRNA by reducing competition for splicing of the major late pre-mRNAs. Preferred Ad vectors containing an E3 deletion that overexpress ADP are GZ1 (SEQ ID NO:3) and GZ3 (SEQ ID NO:4), whose construction and properties are described in the Examples below.

The invention also provides ADP-expressing vectors whose replication is restricted to 25 dividing cells. Any means known to provide such a replication-restricted phenotype may be used. For example, WO 96/40238 describes microbes that preferentially invade tumor cells as well as methods for identifying and isolating bacterial promoters that are selectively activated in tumors. It is also contemplated that expression of one or more vector proteins essential for replication can be placed under the control of the promoter for a cellular gene 30 whose expression is known to be upregulated in neoplastic cells. Examples of such genes include but are not limited to: the breast cancer markers mammaglobin (Watson et al., *Oncogene* 16:817-824, 1998); BRCA1 (Norris et al., *J. Biol. Chem.* 270:22777-22782, 1995) her2/neu (Scott et al., *J. Biol. Chem.* 269:19848-19858, 1994); prostate specific antigen (U.S. Patent 5,698,443); surfactant protein B for lung alveoli (Yan et al., *J. Biol. Chem.* 270:24852-35 24857, 1995); factor VII for liver (Greenberg et al., *Proc. Natl. Acad. Sci. USA* 92:12347-12351, 1995); and survivin for cancer in general (Li et al., *Nature* 396:580-584). Where the

vector is an adenovirus, it is contemplated that such tumor-specific promoters can be substituted for the E4 promoter. Because E4 gene products are essential for Ad replication, placing their expression under the control of a tumor-specific promoter should restrict replication of the vector to tumor cells in which the promoter is activated.

- 5 Another strategy for restricting replication of ADP-expressing Ad vectors to neoplastic cells is exemplified by the KD1 (SEQ ID NO:1), KD2 (SEQ ID NO:13) and KD3 (SEQ ID NO:2) vectors, whose construction and properties are described in the Examples below. This strategy exploits a pre-existing Ad5 mutant in the E1A gene, named *d/1101/1107* (Howe et al., *Proc. Natl. Acad. Sci.*, 87:5883-5887, 1990), also referred to herein as *d/01/07*,
10 and which can only grow well in cancer cells. The role of E1A is to drive cells from the G₀ and G₁ phases of the cell cycle into S-phase. This is achieved by two mechanisms, one involving pRB (and family members), and the other involving p300 and the related protein CBP (DePinho, R.A., *Nature* 391:533-536, 1998). One domain in E1A binds members of the pRB family. pRB normally exists in the cell as a complex with the transcription factor E2F-1
15 and E2F family members (E2F), tethered via E2F to E2F binding sites in promoters of cells expressed in S-phase. Here, pRB acts as a transcriptional co-repressor. E1A binding to pRB relieves this repression, and causes the release of E2F from pRB/E2F complexes. Free E2F then activates promoters of genes expressed in S-phase, e.g. thymidine kinase, ribonucleotide reductase, etc. Another domain in E1A binds the p300/CBP transcription adaptor protein
20 complex. p300/CBP is a transcriptional co-activator that binds many different transcription factors and accordingly is targeted to promoters. p300/CBP has intrinsic histone acetyltransferase activity. E1A binding to p300/CBP is believed to inhibit this histone acetyltransferase activity, allowing acetylation of histones and repression of transcription (Chakravarti et al., *Cell* 96:393-403, 1999; Hamamori et al., *Cell* 96:405-413, 1999).
25 Conceivably, some of the genes that are repressed as a result of E1A interacting with p300/CBP to play a role in blocking the cell cycle, although this is not known. Cancer cells are cycling, so they have free E2F and presumably some p300/CBP-regulated genes are repressed. Consistent with these ideas, E1A must bind both p300/CBP and the pRB family in
30 order to transform primary cells to a constitutively cycling state (Howe et al., *supra*). The mutant *d/01/07* lacks both the p300/CBP- and pRB-binding domains and, as expected, it replicates very poorly in non-dividing "normal" cells or serum-starved cancer cells, but well in growing cancer cells. As described below, the growth of the KD1 and KD3 vectors, which contain the *d/01/07* E1A mutation, is very much better in dividing cancer cells as compared to non-dividing cells. Because the *d/01/07* mutant is completely defective in oncogenic
35 transformation of rat cells (Howe et al., *supra*), vectors according to the invention that contain

this E1A mutation cannot induce cancer in humans (remote as that may be) through an E1A-dependent mechanism.

The invention also includes vectors overexpressing ADP whose replication is restricted to specific tissues by placing expression of one or more proteins essential for 5 replication under the control of a tissue specific promoter and/or a tumor specific promoter. A number of tissue-specific and/or tumor specific promoters have been described in the art. Non-limiting examples include the surfactant protein B promoter, which is only active in cells containing the TTF1 transcription factor (i.e., type II alveolar cells (Yan et al., *supra*)), as described in U.S. Patent 5,466,596 to Breitman et al., which directs gene expression 10 specifically in cells of endothelial lineage; prostate specific antigen which is expressed in prostate cells (Rodriguez et al., *supra*); human telomerase protein (hTERT) promoter (see, e.g., U.S. Patent No. 6,054,575); and human alpha-lactalbumin gene which is expressed in breast cancer cells (Anderson et al., *Gene Therapy* 6:854-864, 1999). Many other tissue-specific, tumor specific, or tissue-preferred enhancer/promoters have been reported (Miller 15 and Whelan, *Human Gene Therapy* 8:803-815, 1997). As exemplified with the surfactant protein B promoter in Examples 6 and 10, vectors expressing tissue-specific promoters would be expected to show tissue specificity in viral replication, viral spreading, cell lysis, and tumor suppression.

Replication of vectors according to the invention can also be controlled by placing 20 one or more genes essential for vector replication under the control of a promoter that is activated by an exogenous inducing agent, such as metals, hormones, antibiotics, and temperature changes. Examples of such inducible promoters include but are not limited to metallothionein promoters, the glucocorticoid promoter, the tetracycline response promoter, and heat shock protein (hsp) promoters such as the hsp 65 and 70 promoters.

The invention also provides compositions comprising a recombinant vector that 25 overexpresses ADP in an amount effective for promoting death of neoplastic cells and a method comprising administering a therapeutically effective amount of the vector to a neoplastic cell in a patient. It is believed the compositions and methods of the present invention are useful for killing neoplastic cells of any origin and include neoplastic cells 30 comprising tumors as well as metastatic neoplastic cells.

It is also contemplated that ADP-expressing viral vectors can be administered to neoplastic cells along with a replication-defective virus that expresses an anti-cancer gene product. For example, many replication-defective E1⁻ Ad vectors for use in cancer therapy are well characterized. A limitation of replication-defective vectors is that they only 35 synthesize the therapeutic protein in the cell they initially infect, they cannot spread to other cells. Also, since the genome does not replicate, transcription can only occur from the input

genomes, and this could be as low as one copy per cell. In contrast, the genome of replication-competent Ad vectors are amplified by about 10^4 in the cell that was initially infected, providing more templates for transcription. More amplification is achieved as the vector spreads to other cells. By combining replication-defective viral vectors expressing an anti-cancer gene product with replication-competent viral vectors described herein, it is expected that the result will be template amplification and rapid spread of both vectors to surrounding cells. For example, with Ad-based vectors, the burst size for each vector should be large, $\sim 10^4$ PFU/cell, so the probability of co-infection of surrounding cells by both vectors will be high. Thus, both the replication-competent and replication-defective vectors should spread simultaneously through the tumor, providing even more effective anti-cancer therapy.

As an alternative method of delivering an anti-cancer gene product with an ADP overexpressing Ad vector, the anti-cancer gene can be engineered into any of the ADP overexpressing replication-competent vectors described herein, in order to provide both the ADP and the anti-cancer function in a single vector. The anti-cancer gene can be engineered 15 into any appropriate location of the vector, as can be easily determined by the skilled artisan. For example, the anti-cancer gene can be engineered into the E3 region.

Expression of the anti-cancer gene product encoded by the replication-defective vector can be under the control of either constitutive, inducible or cell-type specific promoters. The anti-cancer gene product can be any substance that promotes death of a neoplastic cell. The term "gene product" as used herein refers to any biological product or products produced as a result of the biochemical reactions that occur under the control of a gene. The gene product can be, for example, an RNA molecule, a peptide, a protein, or a product produced under the control of an enzyme or other molecule that is the initial product of the gene, i.e., a metabolic product. For example, a gene can first control the synthesis of an RNA molecule which is translated by the action of ribosomes into a prodrug converting enzyme which converts a nontoxic prodrug administered to a cancer patient to a cell-killing agent; the RNA molecule, enzyme, and the cell-killing agent generated by the enzyme are all gene products as the term is used here. Examples of anti-cancer gene products include but are not limited to cell-killing agents such as apoptosis-promoting agents and toxins; prodrug 25 converting enzymes; angiogenesis inhibitors; and immunoregulatory molecules and antigens capable of stimulating an immune response, humoral and/or cellular, against the neoplastic cell.

Apoptosis-promoting agents include but are not limited to the pro-apoptotic members of the BCL-2 family such as BAX, BAD, BID and BIK, as well as antisense molecules which 35 block expression of anti-apoptotic members of the BCL-2 family. Examples of immunoregulatory molecules are cytokines such as tumor necrosis factor, Fas/Apo1/CD95

ligand, tumor necrosis factor related apoptosis inducing ligand, interleukins, macrophage activating factor and interferon γ . Angiogenesis inhibitors include but are not limited to endostatin and angiostatin. Toxins include but are not limited to tumor necrosis factor, lymphotoxin, the plant toxin ricin, which is not toxic to humans due to the lack of ricin receptors in animal cells, and the toxic subunit of bacterial toxins. Examples of pro-drug converting enzymes and pro-drug combinations are described in WO 96/40238 and include thymidine kinase and acyclovir or gancyclovir; and bacterial cytosine deaminase and 5-fluorocytosine.

The therapeutic or pharmaceutical compositions of the present invention can be administered by any suitable route known in the art including for example by direct injection into a tumor or by other injection routes such as intravenous, subcutaneous, intramuscular, transdermal, intrathecal and intracerebral. Administration can be either rapid as by injection or over a period of time as by slow infusion or administration of slow release formulation. For treating tissues in the central nervous system, administration can be by injection or infusion into the cerebrospinal fluid (CSF). When it is intended that a recombinant vector of the invention be administered to cells in the central nervous system, administration can be with one or more agents capable of promoting penetration of the vector across the blood-brain barrier. Preferably, vectors of the invention are administered with a carrier such as liposomes or polymers containing a targeting moiety to limit delivery of the vector to targeted cells.

Examples of targeting moieties include but are not limited to antibodies, ligands or receptors to specific cell surface molecules.

Compositions according to the invention can be employed in the form of pharmaceutical preparations. Such preparations are made in a manner well known in the pharmaceutical art. One preferred preparation utilizes a vehicle of physiological saline solution, but it is contemplated that other pharmaceutically acceptable carriers such as physiological concentrations of other non-toxic salts, five percent aqueous glucose solution, sterile water or the like may also be used. It may also be desirable that a suitable buffer be present in the composition. Such solutions can, if desired, be lyophilized and stored in a sterile ampoule ready for reconstitution by the addition of sterile water for ready injection.

The primary solvent can be aqueous or alternatively non-aqueous.

The carrier can also contain other pharmaceutically-acceptable excipients for modifying or maintaining the pH, osmolarity, viscosity, clarity, color, sterility, stability, rate of dissolution, or odor of the formulation. Similarly, the carrier may contain still other pharmaceutically-acceptable excipients for modifying or maintaining release or absorption or penetration across the blood-brain barrier. Such excipients are those substances usually and customarily employed to formulate dosages for parenteral administration in either unit dosage

or multi-dose form or for direct infusion into the cerebrospinal fluid by continuous or periodic infusion.

It is also contemplated that certain formulations containing ADP-expressing vectors are to be administered orally. Such formulations are preferably encapsulated and formulated with suitable carriers in solid dosage forms. Some examples of suitable carriers, excipients, and diluents include lactose, dextrose, sucrose, sorbitol, mannitol, starches, gum acacia, calcium phosphate, alginates, calcium silicate, microcrystalline cellulose, polyvinylpyrrolidone, cellulose, gelatin, syrup, methyl cellulose, methyl- and propylhydroxybenzoates, talc, magnesium, stearate, water, mineral oil, and the like. The formulations can additionally include lubricating agents, wetting agents, emulsifying and suspending agents, preserving agents, sweetening agents or flavoring agents. The compositions may be formulated so as to provide rapid, sustained, or delayed release of the active ingredients after administration to the patient by employing procedures well known in the art. The formulations can also contain substances that diminish proteolytic degradation and promote absorption such as, for example, surface active agents.

The specific dose is calculated according to the approximate body weight or body surface area of the patient or the volume of body space to be occupied. The dose will also be calculated dependent upon the particular route of administration selected. Further refinement of the calculations necessary to determine the appropriate dosage for treatment is routinely made by those of ordinary skill in the art. Such calculations can be made without undue experimentation by one skilled in the art. Exact dosages are determined in conjunction with standard dose-response studies. It will be understood that the amount of the composition actually administered will be determined by a practitioner, in the light of the relevant circumstances including the condition or conditions to be treated, the choice of composition to be administered, the age, weight, and response of the individual patient, the severity of the patient's symptoms, and the chosen route of administration. Dose administration can be repeated depending upon the pharmacokinetic parameters of the dosage formulation and the route of administration used.

The invention also contemplates passively immunizing patients who have been treated with a viral vector overexpressing ADP. Passive immunization can include administering to the patient antiserum raised against the viral vector, or gamma-globulin or vector-specific purified polyclonal or monoclonal antibodies isolated from the antiserum. Preferably, the patient is passively immunized after a time period sufficient for the viral vector to replicate in and spread through the tumor.

Preferred embodiments of the invention are described in the following examples. Other embodiments within the scope of the claims herein will be apparent to one skilled in the

art from consideration of the specification or practice of the invention as disclosed herein. It is intended that the specification, together with the examples, be considered exemplary only, with the scope and spirit of the invention being indicated by the claims which follow the examples.

5

Example 1

This example illustrates the construction and characterization of the KD1 and KD3 anti-cancer vectors.

To construct KD1, the inventors deleted the entire E3 region of a unique plasmid, leaving behind only a unique PacI site for cloning. The starting plasmid was pCRII, purchased from Invitrogen, containing the Ad5 BamHIA fragment having a deletion of all the E3 genes; the E3 deletion is identical to that for KD1 and GZ3, the sequences of which are given in SEQ ID NO:1 and SEQ ID NO:4, respectively. The ADP gene from Ad5 was cloned into the PacI site, then built into the E3 region of the genome of the Ad5 E1A mutant named *d/01/07*. This was done by co-transfected into human embryonic kidney 293 cells the aforementioned BamHIA fragment containing the ADP gene together with the overlapping EcoRIA restriction fragment obtained from *d/01/07*. Complete viral genomes are formed within the cell by overlap recombination between the Ad sequences in the BamHIA fragment in the plasmid and the EcoRIA fragment. KD3 was constructed in the same way except the E3 gene for the 12.5K protein was retained in the starting plasmid. A vector named KD2, which marginally overexpress ADP, was also prepared. Plaques of each recombinant Ad were picked, screened, purified, expanded into CsCl-banded stocks, sequenced, titered, and characterized. GZ1 and GZ3 are Ad vectors that are identical to KD1 and KD3, respectively, except that GZ1 and GZ3 have wild-type E1A sequences as found in Ad5 or in the Ad5 mutant *d/309*. GZ1 and GZ3 were constructed as described for KD1 and KD3 except that the EcoRIA fragment of Ad5 was used for GZ1 and GZ3.

KD1 and KD3 were characterized in cell culture by infecting the human A549 lung carcinoma cell line with high titer ($1\text{-}8 \times 10^{10}$ plaque forming units [PFU] per ml) virus stocks of one of these recombinant vectors, or with one of the control viruses *d/01/07*, *d/309*, *d/327*, and Ad5 (wt). Fifty PFU per cell were used for each virus. The descriptions of these viruses as well as some other viruses used in these examples are presented in Table 1.

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Table 1: Description of mutations in viruses:

us	RNA REGION			E4
	E1	VA	E3	
<u>10I/1107</u>	<i>dI101: deletion of Ad5 bp 569-634</i> <i>dI107: deletion of Ad5 bp 890-928</i>	From <i>dI309</i> deletion of Ad5 bp 10594-10595	From <i>dI309</i> deletion of Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin	wild type
<u>1</u>	<i>dI101: deletion of Ad5 bp 569-634</i> <i>dI107: deletion of Ad5 bp 890-928</i>	From <i>dI309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 27858-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	wild type
<u>2</u>	<i>dI101: deletion of Ad5 bp 569-634</i> <i>dI107: deletion of Ad5 bp 890-928</i>	From <i>dI309</i> deletion of Ad5 bp 10594-10595	<i>dI309</i> background, gp19K mutated deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin; deletion of Ad5 bp 28788-28789, insert TTAATTAA	wild type
<u>3</u>	<i>dI101: deletion of Ad5 bp 569-634</i> <i>dI107: deletion of Ad5 bp 890-928</i>	From <i>dI309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	wild type
<u>1</u>	wt	wild type	deletion of Ad5 bp 27858-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAATTAAAGG	wild type

	wild type	wild type	wild type	wild type
01/1107-	<i>d11101:</i> deletion of Ad5 bp 569-634 <i>d11107:</i> deletion of Ad5 bp 890-928	From <i>d1309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	From <i>d1309</i> deletion of Ad5 bp 28597-28602; deletion-substitution Ad5 bp 3005-30750, insert 642 bp DNA of unknown origin
-SPB	<i>d11101:</i> deletion of Ad5 bp 569-634 <i>d11107:</i> deletion of Ad5 bp 890-928	From <i>d1309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 27848-2760, TAA inserted; deletion of Ad5 bp 27982-28134; deletion of Ad5 bp 28395-29397, insert CCCTTAATTAAA; deletion of Ad5 bp 29783-30883, insert TTAAATTAAAGG	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by Bst1 1071 sites
-SPB	<i>d11101:</i> deletion of Ad5 bp 569-634 <i>d11107:</i> deletion of Ad5 bp 890-928	From <i>d1309</i> deletion of Ad5 bp 10594-10595	deletion of Ad5 bp 28598-29397; deletion of Ad5 bp 29783-30469	E4 promoter deletion-substitution: deletion of Ad5 bp 35623-35775, insert SP-B 500 promoter flanked by Bst1 1071 sites

Using a polymerase chain reaction (PCR)-based protocol, an in-frame stop codon was introduced into the gene for the E3-gp19K protein in the E3 region of the Ad5 mutant dI309 (Jones and Shenk, *Cell* 17:683-689, 1979). The mutagenesis was conducted using a SmaI-Bst1107I fragment, nucleotides 28,390 to 29,012 in the Ad5 genome, which was then substituted for the equivalent fragment in dI309. dI01/07 is the parent for KD1 and KD3. In turn, the Ad5 mutant named dI309 is the parent of dI01/07, i.e. dI309 is identical to dI01/07 except that dI309 does not have the E1A mutation. Both dI01/07 and dI309 have deletions of the genes for the E3 RID α , RID β and 14.7K proteins but retain the gene for ADP. The Ad5 mutant dI327 has wild-type E1A, it lacks the gene for ADP, and its lacks all other E3 genes except the one for the 12.5K protein.

At 24 and 36 hours post-infection (h p.i.), proteins were extracted from the A549 cells and analyzed for ADP by immunoblot using a rabbit antiserum against ADP (Tollefson et al., *J. Virol.* 66:3633-3642, 1992). The results are shown in Figure 2. Much more ADP was detected at 24 and 36 h p.i. in KD1- and KD3-infected cells than in cells infected with dI01/07. Also, much more ADP was synthesized by GZ1 and GZ3 than dI309 or the other viruses. Most importantly, KD1, KD3, GZ1, and GZ3 expressed much more ADP at 24 h p.i. than did dI01/07 or dI309 (Fig. 2). This result is consistent with an observation discussed below that the cells infected with KD1, KD3, GZ1, or GZ3 lyse faster, and that these viruses spread from cell to cell faster than dI01/07 or dI309. It is noteworthy that KD1, KD3, GZ1, and GZ3 express much more ADP at 24 and 36 h p.i. than the Ad5 mutant dI1520 (Fig. 2); dI1520 is the original name given to ONYX-015 (Heise et al., *Nature Medicine* 3:639-645, 1997). As expected, no ADP was detected in cells infected with pm734.1 (Fig. 2), a mutant that lacks amino acids 1 to 48 in ADP (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). Expression of the E1A proteins by dI01/07, KD1, KD2, and KD3 was slightly less than by Ad5, dI309, or dI327, and as expected from the dI01/07 deletion, the proteins were smaller (Fig. 3A). dI327 is isogenic with dI324 (Thimmappaya et al., 1982 *Cell* 31:543-51, 1983), and it lacks the gene for ADP and all other E3 proteins except the 12.5K protein.

The amount of ADP detected in the KD1 and KD3 infected cells is significantly higher than the amount detected in the dI309 infected cells (Fig. 2). If one takes into consideration the fact that the viruses with the E1A mutation replicate somewhat slower, as evidenced in by the delayed appearance of the late proteins (Fig. 3B), it is clear that KD1 and KD3 express much more ADP per viral genome present in the cell than dI309. This finding is supported by the fact that when A549 cells are coinfecte with a virus containing the E1A mutation and dI327, which lacks ADP but has wild-type E1A, the replication rates of the E1A mutant viruses speed up, as indicated by earlier appearance of late proteins (compare Figs. 3B

and 3D). Thus, *dl327* complements the E1A mutation. In conclusion, these experiments demonstrate that ADP is dramatically overexpressed by KD1, KD3, GZ1, and GZ3. ADP is marginally overexpressed by KD2 (not shown).

Example 2

5 This example illustrates that KD1 and KD3 lyse cells more rapidly and spread from cell-to cell faster than other adenoviruses.

The ability of KD1 and KD3 to lyse cells was examined by a trypan blue exclusion cell viability assay which was performed essentially as described by Tollefson et al., *J. Virol.* 70:2296-2306, 1996. In brief, A549 cells were mock-infected or infected with 20 PFU/cell of 10 KD1, KD3, *dl01/07*, *dl327* or *dl309*. At various days p.i., the number of viable cells was determined using a hemocytometer (600 to 1000 cells were counted per time point) and the results are shown in Fig. 4.

Only 25% of the KD1-infected cells and 9% of the KD3-infected cells were alive at 5 days p.i. as compared to 44% of cells infected with *dl01/07*, which has the same E1A 15 mutation as KD1 and KD3. The KD1 and KD3 vectors also lysed cells faster than *dl309*, which has a wild-type E1A region. When infected with *dl327* (ADP⁻, E1A⁺), 94% of the cells were alive after 5 days. When cell lysis was estimated by release of lactate dehydrogenase, KD1 and KD3 once again lysed cells faster than *dl01/07* and *dl309*, and *dl327* caused little 20 cell lysis (data not shown). Thus, ADP is required for efficient cell lysis, and over-expression of ADP increases the rate of cell lysis.

As another means to measure cell lysis and to examine virus replication in cancer cells, separate groups of A549 cells were infected with 20 PFU/cell of KD1, KD3, *dl01/07*, or *dl309* and the amount of intracellular and extracellular virus was determined by plaque assay on A549 cells. At 2 days p.i., the total amount of virus formed in each group was similar, 2-4 25 x 10⁸ PFU/ml, indicating that replication of all the viruses is similar. However, when the ratio of extracellular to intracellular virus was calculated, the value for KD1 and KD3 was 2-3 logs higher than for Ad5, *dl309*, or *dl01/07* (data not shown). Thus, virus is released much more rapidly from cells infected with KD1 and KD3, which overexpress ADP, than with viruses expressing wild-type amounts of ADP.

30 The ability of KD1 and KD3 to spread from cell-to-cell was measured in a "cell spreading" assay. In this assay monolayers of A549 cells in a 48 well culture dish were mock-infected or infected with 10³, 10², 10¹, 10⁰, or 10 PFU/cell of *dl327*, *dl309*, Ad5, *dl01/07*, KD1 or KD3. At low PFU/cell, the viruses must go through two or three rounds of replication in order to infect every cell in the monolayer. At 1.0 and 10 PFU/cell, the 35 monolayer should be destroyed by the virus that initially infected the cells. To assess the

amount of spread in the monolayers by 7 days p.i., crystal violet, which stains live cells but not dead cells, was added to the monolayers. The results are shown in Fig. 5.

Remarkably, at 7 days p.i., the monolayer was virtually eliminated by KD1 and KD3 at 10^{-3} PFU/cell, whereas 1.0 PFU/cell was required with *d/01/07*, *d/309* and Ad5. This result
5 attests to the potency of ADP in mediating cell lysis and virus spread in A549 cells. KD1 and KD3 are also more effective than *d/01/07* in killing other types of human cancer cell lines (most purchased from the American Type Culture Collection [ATCC]) as determined in this cell spreading assay. KD1 and/or KD3 killed HeLa (cervical carcinoma), DU145 (prostate),
10 and pC3 (prostate) cells at 10^{-2} PFU/cell, ME-180 (cervix) and Hep3B (liver) at 10^{-1} PFU/cell, and U118 (glioblastoma) and U373 (glioblastoma) at 10 PFU/cell. From 10- to 100-fold
more *d/01/07* was required to kill these cells (data not shown). These results indicate that
KD1 and KD3 may be effective against many types of cancer.

An important aspect of the finding that ADP overexpressing vectors lyse cells at very low multiplicities of infection is that the multiplicity of infection in human tumors is likely to
15 be low at sites distal to the sight of vector injection or distal to blood vessels that carry the vector to the tumor. Thus, ADP overexpressing vectors have an advantage over vectors that express less ADP or no ADP at all.

Example 3

This example illustrates that KD1 and KD3 replicate poorly in non-growing non-cancerous cells. The replication phenotype of KD1 and KD3 was evaluated using "normal"
20 HEL-299 human fibroblast cells, either growing in 10% serum or rendered quiescent using 0.1% serum. All Ads should replicate well in growing cells, but viruses with the *d/01/07* E1A mutation should do poorly in quiescent cells because E1A is required to drive them out of G₀. *d/309*, which has wild-type E1A, should replicate well in both growing and growth-arrested
25 cells.

Cells were infected with 100 PFU/cell of KD1, KD3, *d/01/07*, or *d/309*. At different days p.i., virus was extracted and titered. In 10% serum, KD1, KD3, and *d/01/07* replicated well, reaching titers of 10^6 - 10^7 PFU/ml, only slightly less than *d/309* (Fig. 6). However, in quiescent cells, replication of KD1, KD3, and *d/01/07* was 1.5-2 logs lower than in growing cells, ranging from 10^4 to 2×10^5 PFU/ml. The titer of *d/309* reached 10^7 PFU/ml, nearly the level achieved in growing cells. At 10 days p.i., quiescent HEL-299 cell monolayers infected with 100 PFU/cell of KD1, KD3, or *d/01/07* were intact, whereas those infected with *d/309* or
30 *d/327*, which have wild-type E1A, showed strong typical Ad cytopathic effect indicative of cell death (data not shown). Thus, replication of KD1 and KD3 is severely restricted to
35 growing cell lines.

The restriction associated with the *dl*01/07 E1A mutation was also tested in primary human cells (purchased from Clonetics) growing as monolayers. Bronchial epithelial cells (Fig. 7) and small airway epithelial cells were not killed by 10 PFU/cell of KD1, KD3, or *dl*01/07 at 5 days p.i., whereas they were killed by 10 PFU/cell of *dl*309 or *dl*327 (data not shown). Lung endothelial cells also were not killed after 10 days by KD1, KD3, or *dl*01/07 at 10 PFU/cell, but they were killed by 1 PFU/cell of *dl*309. These monolayers were subconfluent when initially infected, then grew to confluence. The exciting result here is that although these primary cells were growing, they did not support replication in this time frame and were not killed by KD1 or KD3. Thus, it is believed these vectors will be restricted to cancerous cells, and will have little to no effect on cells such as basal cells that are normally dividing in the body. In addition, it is unlikely that KD1 and KD3 will affect dividing leukocytes because such cells are poorly infected by Ad.

In summary, the above experiments demonstrate that KD1 and KD3 lyse cancer cells, spread from cell-to-cell rapidly, and replicate poorly in quiescent and non-cancerous cells. 15 These properties should make them useful in anti-cancer therapy.

Example 4

This example illustrates that KD1 and KD3 inhibit the growth of human tumors in an animal model.

We could not evaluate mouse or rat tumors in normal mice or rats because they are totally non-permissive. Human cancer cell lines growing in nude mice have been used by Onyx Pharmaceuticals (Richmond, CA) to evaluate the efficacy of ONYX-015, an Ad vector lacking expression of the E1B 55 kDa protein (Heise et al., *Nature Med.* 3:639-645, 1997). We have found that A549 cells, which were used in many of our cell culture studies, form excellent rapidly growing solid tumors when injected subcutaneously into nude mice. The 25 average tumor reaches ca. 500 µl in four weeks, and is encapsulated, vascularized, and attached to the mouse skin (usually) or muscle.

Nude mice were inoculated into each hind flank with 2×10^7 A549 cells. After 1 week tumors had formed, ranging in size from about 20 µl to 50 µl. Individual tumors were injected three days later, and at subsequent weeks for 4 weeks (total of 5 injections), with 50 µl of buffer or 50 µl of buffer containing 5×10^7 PFU of *dl*309, *dl*01/07, KD1, KD3, or *pm*734.1, with a total virus dose per tumor of 3×10^8 PFU. The mutant *pm*734.1 lacks ADP activity due to two nonsense mutations in the gene for ADP, but all other Ad proteins are expected to be synthesized at wild-type levels (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). The efficacy of each virus (or buffer) was tested on six tumors. At weekly intervals, the 35 length (L) and width (W) of tumors were measured using a Mitutoyo digital caliper. Tumor

volumes were calculated by multiplying L x W x W/2. This value was divided by the tumor volume at the time of the initial virus injection, the fold-increase in tumor growth was calculated, and the average for the six tumors was graphed.

As shown in Fig. 8A, tumors that received buffer continued to grow, increasing about 5 14-fold by 5 weeks. In contrast, tumors injected with *dl309*, which expresses normal amounts of ADP and lacks the E3 RID and 14.7K and proteins, only grew about 2.5-fold by 5 weeks. With *pm734.1*, which lacks ADP, the tumors grew as well as those that received buffer. Thus, *dl309* markedly decreases the rate of tumor growth, and ADP is required for this decrease. Tumors inoculated with *dl01/07* grew about 8-fold over 5 weeks. Since *dl01/07* is 10 identical to *dl309* except for the E1A mutation, this result indicates that the E1A mutation significantly reduces the ability of Ad to prevent growth of the tumors. This effect is probably due to a reduction in virus replication in the tumors resulting in lower ADP expression, but it could also reflect other properties of E1A in the tumor cells, e.g. the inability of the mutant E1A proteins to induce apoptosis. Most importantly, tumors 15 inoculated with KD1 or KD3 only grew about 2.5-fold. Thus, the overexpression of ADP by KD1 and KD3 allows KD1 and KD3 to reduce tumor growth to a rate markedly slower than *dl01/07* (their parental control virus), and even to a rate similar to that of *dl309*.

The finding that KD1 and KD3 are as effective as wild-type Ad (i.e. *dl309*) in reducing the rate of A549 tumor growth is highly significant in the context of cancer 20 treatment, inasmuch as KD1 and KD3 are restricted to cancer cells whereas wild-type Ad does not have such a restriction.

The tumors in Fig. 8A received five injections of vectors, but only one dose of vector, in this case 5×10^8 of each of KD3 or GZ3, is sufficient to significantly reduce the rate of A549 tumor growth (Fig. 8B).

We have also found that KD1 and KD3 reduce the rate of growth in nude mice of a 25 human liver cancer cell line, Hep3B cells. These cells form rapidly growing tumors that are highly vascularized. Nude mice were inoculated into each hind flank with 1×10^7 of Hep3B cells. After tumors reached about 100 μ l, they were injected twice per week for 3 weeks with 50 μ l of buffer or 5×10^7 PFU of KD1, KD3, or *dl309*. There were typically 8-10 tumors per 30 test virus. The tumor sizes were measured and the fold increase in size at 0 to 3.5 following the initial virus injection was graphed as described above for the A549 tumors. Tumors that received buffer alone grew 9-fold over 3 weeks and were projected to grow about 12-fold over 3.5 weeks (after 3 weeks the mice had to be sacrificed because the tumors were becoming too large) (Fig. 9). Tumors that received KD1 or KD3 grew about 4-fold, 35 establishing that KD1 and KD3 reduce the growth of Hep3B tumors in nude mice. Tumors

that were injected with *dl309* grew 2-fold (Fig. 9). The finding that KD1 and KD3 were somewhat less effective than *dl309* is probably due to the fact that they do not grow as well as *dl309* in Hep3B cells, as indicated by a cell spread assay in culture (data not shown). In any case, the important points are that KD1 and KD3 are effective against the Hep3B tumors, and 5 that they contain the E1A mutation that limits their replication to cancer cells.

These results point to the potency of ADP as an anti-tumor agent when expressed in an Ad vector. It is highly probable that KD1 and KD3 will provide significant clinical benefit when used to infect tumors growing in humans.

Example 5

10 This example illustrates the use of replication-defective Ad vectors in combination with KD1 or KD3.

It is well established that replication-competent (RC) viruses complement replication-defective (RD) mutants. That is, when the same cell is infected, the competent virus will supply the protein(s) that cannot be made from the mutant genome, and both viruses will 15 grow. To test the ability of KD1 and KD3 to complement RD viruses, two RD vectors expressing β-galactosidase were constructed. The first, named Ad-β-gal, has a cDNA encoding β-gal under the control of the Rous Sarcoma Virus promoter substituted for the deleted E1 region. Ad-β-gal also has the E3 region deleted, including the gene for ADP. The second, named Ad-β-gal/FasL is identical to Ad-β-gal, except that it also expresses murine 20 FasL from the human cytomegalovirus promoter/enhancer. These vectors were constructed by overlap recombination in human 293 cells that constitutively express the Ad E1A and E1B genes and complement replication of the E1-minus vectors.

These RD vectors should infect and express β-gal in A549 cells, but should not replicate because the E1A proteins are lacking. However, the vectors should replicate when 25 cells are co-infected with RC Ads. To prove this, A549 cells were infected with 10 PFU/cell of Ad-β-gal alone, or with 10 PFU/cell of Ad-β-gal plus 10 PFU/cell of KD1, KD3, *dl01/07*, *dl309*, or *dl327*. At 2 days p.i., virus was extracted and Ad-β-gal titers determined by β-gal expression in A549 cells. The yields are shown in Table 2 below.

Table 2

Virus	Yield (blue plaques per ml)
Ad-β-gal	1×10^2
Ad-β-gal + KD1	2×10^5
Ad-β-gal + KD3	3×10^5
Ad-β-gal + dl01/07	4×10^4
Ad-β-gal + dl309	3×10^5
Ad-β-gal + dl327	3.0×10^5

The data in Table 2 indicate that the complementing viruses increased the yield of Ad-β-gal by about 10^3 .

5 A key feature of KD1 and KD3 is that they spread from cell-to-cell faster than other Ads. Accordingly, they should complement the spread of Ad-β-gal. To test this, an infectious center assay was conducted. A549 cells were infected with Ad-β-gal plus KD1, KD3, or dl01/07. After 2 h, cells were collected, diluted, and seeded onto monolayers of fresh A549 cells. After 4 days, the cells were stained with X-gal and the results are shown in
10 Fig. 10.

With Ad-β-gal alone, only the originally infected cell (before seeding) should be stained, and the vector should not spread to other cells on the seeded monolayer. This was indeed the case. In monolayers seeded with A549 cells infected with Ad-β-gal alone (dish shown in the top left of Fig. 10A) contained a number of individual blue cells (not visible in
15 the print); examples are shown in the enlarged view Fig. 10B. However, when the monolayers were seeded with A549 cells coinfecte with Ad-β-gal and KD1 or KD3, there were numerous "comets" of blue cells (Fig. 10A). Each comet represents Ad-β-gal which has spread from one initially-infected cell. Most of the cells within a comet were stained with X-gal (Fig. 10C). Comets were also observed with dl01/07, but not to the extent of KD1 and
20 KD3 (Fig. 10A). With dl327 (ADP), there was little spread from the originally infected cell (data not shown). In summary, KD1 and KD3 not only complement the replication of Ad-β-gal, they also enhance its rapid spread.

It is expected that KD1 and KD3 will also complement and enhance the spread of RD vectors expressing anti-cancer therapeutic gene products, and this expectation can be readily

verified using the Ad- β -gal/FasL in replication and infectious center assays as described above.

KD1 and KD3 not only complement the replication of RD vectors in cell culture, they also do so in Hep3B tumors growing in the hind flanks of nude mice. The RD vector used was AdLuc, an Ad that lacks the E1 and E3 regions, and has inserted into the E1 region an expression cassette where the firefly luciferase gene is expressed from the Rous Sarcoma Virus promoter (Harrod et al., *Human Gene Therapy* 9:1885-1898, 1998). The Hep3B tumors were injected with 1×10^7 PFU of AdLuc plus buffer, or 1×10^7 PFU of AdLuc plus 5×10^7 PFU of KD1, KD3, dI01/07, or dI309. After 2 weeks, mice were sacrificed and tumors excised. Proteins were extracted from the tumors and luciferase activity determined using a luminometer. The luciferase counts per tumor were 6,800 for AdLuc plus buffer, 113,500 for KD1, and 146,900 for KD3 (Fig. 11). Thus, KD3 and KD1 respectively caused a 22-fold and 17-fold increase in luciferase activity. This increase could be due to elevated synthesis of luciferase in cells that were initially coinjected the AdLuc and KD1 or KD3, and it could also be due to spread of AdLuc from cell to cell in the tumor as mediated by KD1 or KD3.

In summary, infecting a tumor with a replication-competent ADP-overexpressing vector according to the invention together with a RD vector expressing an anti-cancer gene product should greatly increase the amount of anti-cancer protein synthesized in the tumor thereby increasing the ability of the replication-defective vector to promote destruction of the tumor.

Example 6

This example illustrates the construction and characterization of a recombinant Ad vector according to the invention which is replication-restricted to cancerous type II alveolar cells.

As demonstrated above, the dI01/07 mutation in KD1 and KD3 limits growth of these vectors to cancer cells. To further restrict their replication phenotype, the E4 promoter in each virus was deleted and replaced by the surfactant protein B (SPB) promoter to produce vectors named KD1-SPB (SEQ ID NO:14), KD3-SPB (SEQ ID NO:15), and dI01/07-SPB (SEQ ID NO:16). The SPB promoter is only active in cells containing the TTF1 transcription factor, which has thus far been found primarily in type II alveolar cells of the human lung (Lazzaro et al., *Development* 113:1093-1104, 1991). Thus, KD1-SPB, KD3-SPB, and dI01/07-SPB should be severely restricted to cancerous type II alveolar cells of the human lung. Many lung cancers are of this type.

The KD1-SPB and KD3-SPB vectors were prepared as follows. The E4 promoter is located at the right end of the Ad genome (Fig. 1). Using a pCRII-based plasmid (Invitrogen)

containing the Ad5 DNA sequences from the BamHI site (59 map units) to the right hand end of the genome, and using and a PCR-based protocol, nearly all the transcription factor binding sites were deleted from the E4 promoter Ad5 base pairs 35,623 to 35,775 and replaced with a 500 base pair fragment containing the SPB promoter (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). The final plasmids contain the E4-SPB substitution in the E4 region and the dI01/07, KD1, or KD3 versions of the E3 region, respectively, for the viruses dI01/07-SPB, KD1-SPB, and KD3-SPB. These plasmids were co-transfected into 293 cells with a fragment containing the left portion of the genome of dI01/07, and plaques were allowed to develop. Plaques were screened for the expected features, purified, then expanded into a stock.

The A549-TTF1 cell line was developed in order to test the prediction that replication of dI01/07-SPB, KD1-SPB, and KD3-SPB would be restricted to cancerous cells expressing the TTF1 transcription factor. These cells were co-transfected with two plasmids, one in which TTF1 is expressed from the CMV promoter, and the other coding for resistance to neomycin. Resistant clones were isolated and shown to express TTF1 activity as determined by transient transfection with a plasmid expressing chloramphenicol acetyltransferase from the TTF1-requiring surfactant protein C promoter.

KD1-SPB and KD1 were subjected to a standard plaque development assay on A549-TTF1 cells and parental A549 cells. The results are shown in Fig. 12. With KD1-SPB on A549 cells, plaques were not visible after 8 days, only about 4% of the final number of plaques were seen after 10 days, and about 50% of final plaques were seen after 12 days. With KD1-SPB on A549-TTF1 cells, plaques were visible after 6 days, and about 60% of plaques were seen after 10 days. Thus, as expected, KD1-SPB grew significantly faster on the cells containing TTF1. KD1 formed plaques more quickly than KD1-SPB on both A549 and A549-TTF1 cells, indicating that the E4 promoter-SPB substitution is not as effective as the wild-type E4 promoter in inducing Ad replication. However, this difference between KD1-SPB and KD1 on A549-TTF1 cells is tolerable, with KD1-SPB delayed only about 1 day. Curiously, the final titer obtained for all virus stocks by day 16 was similar, indicating that A549 cells may contain a very small amount of endogenous TTF1 activity. It is predicted that KD3-SPB and dI01/07-SPB will behave similarly to KD1-SPB when grown in A549-TTF1 cells and A549 cells.

The restriction of KD1-SPB to cells containing TTF1 was further examined in a cell spread assay using H441 cells, a TTF1-expressing human pulmonary adenocarcinoma cell line (Yan et al., *supra*), and Hep3B cells, a liver cancer cell line not expected to express TTF1. Culture dish wells containing H441 or Hep3B cells were infected with KD1-SPB or KD1 at multiplicities ranging from 10 to 10^4 PFU/cell. The H441 and Hep3B cells were

stained with crystal violet at 5 days and 8 days p.i., respectively. KD1-SPB and KD1 grew and spread equally well on H441 cells, causing destruction of the monolayer at 10^{-1} PFU per cell (Fig. 13). (Some of the H441 monolayer has peeled off in the well with KD1-SPB at 10^{-2} PFU per cell, and in the wells with KD1 and KD1-SPB at 10^{-4} PFU per cell; this occasionally occurs in cell spread assays, and it does not reflect virus infection). With Hep3B cells, KD1 grew and spread very much better than KD1-SPB, with 10^{-2} PFU per cell of KD1 causing more destruction of the monolayer as 1.0 PFU per cell of KD1-SPB (Fig. 13).

In summary, this example demonstrates that a replication-competent Ad, which replicates well on cells expressing the appropriate transcription factor, can be constructed with a tissue-specific promoter substituted in place of the E4 promoter. This methodology should be applicable to many other tissue specific and cell type specific promoters. One possibility would be a liver-specific promoter. Another possibility would be to use the E2F promoter, or another promoter with E2F sites, inasmuch as that promoter would be active only in cells such as cancer cells that have free E2F. A third possibility would be to use a regulatable promoter, e.g. the synthetic tetracycline response promoter (Massie et al., *J. Virol.* 72:2289-2296, 1998), where the activity of the promoter is controlled by the level of tetracycline or a tetracyclin analog in the patient.

Example 7

This example illustrates the construction and characterization of vectors which overexpress ADP and are not replication restricted.

As demonstrated above, the *d101/07* E1A mutation in KD1 and KD3 is attenuating, inhibiting growth in non-dividing and even in dividing primary human epithelial and endothelial cells. Ads with this mutation are able to replicate well in dividing cancer cells. However, replication of such E1A mutants is not as efficient as, e.g. *d1309* which has a wild-type E1A gene. For instance, the rate of replication of *d101/07*, as determined by the rate at which plaques develop, is reduced such that *d101/07* plaques appear one day later than those of *d1309* (data not shown). This delay is due in part to a delay in expression of Ad late genes (see Fig. 3). The idea that the *d101/07* mutation retards the rate of replication in A549 cells is further supported by the data in Fig. 8A, where *d101/07* did not prevent tumor growth nearly as well as *d1309*. Despite this negative effect of the *d101/07* E1A mutation, there are theoretical and practical aspects of having this mutation in the KD1 and KD3 vectors, as has been discussed. Nevertheless, one can easily imagine scenarios (e.g. patients with terminal cancer) where the ability of an Ad vector to destroy the tumor supercedes the requirement that the vector be totally restricted to tumor cells. In such cases, it would be advantageous to have vectors similar to KD1 and KD3, but with the wild-type E1A gene. The rates at which such

vectors express their genes, lyse cells, and spread from cell to cell should be higher than those of KD1 and KD3. Such vectors might cause some damage to non-cancerous cells and tissue, but this is also true for other modes of anti-cancer treatment such as surgery, chemotherapy, and radiation therapy.

5 In light of these considerations, vectors named GZ1 and GZ3 have been constructed that are identical to KD1 and KD3, respectively, except they have a wild-type E1A region. These vectors were constructed by overlap recombination in A549 cells. The left hand fragment contained the wild-type E1A region of Ad5, and the right end fragment contained the E3 modifications of KD1 or KD3. Plaques were picked, analyzed for the expected
10 genotype, plaque-purified, and expanded into CsCl-banded stocks. The titers of these stocks on A549 cells were 2.9×10^{10} PFU/ml for GZ1 and 1.6×10^{11} PFU/ml for GZ3. Thus, these vectors can be grown into high titer stocks comparable to wild-type Ad. The GZ1 and GZ3 plaques are larger and appear much sooner than the plaques for *dl*309. Large rapidly-
15 appearing plaques reflect the ability of Ad to lyse cells and spread from cell-to-cell (Tollefson et al., *J. Virol.* 70:2296-2306, 1996; Tollefson et al., *Virology* 220:152-162, 1996), and this property, as discussed, is due to the function of ADP.

The rate of plaque appearance can be quantitated in a plaque development assay (Tollefson et al., *supra*). Here, a typical plaque assay is performed, and the plaques observed on subsequent days of the assay are calculated as a percentage of the number of plaques
20 observed at the end of the plaque assay. As shown in Fig. 14, after 4 days of plaque assay on A549 cells, GZ1 and GZ3 had 48% and 34%, respectively, of the final number of plaques, whereas *dl*309 had only 1%. It is very unusual in Ad plaque assays in A549 cells for plaques to appear after only 4 days. These large plaques reflect the overexpression of ADP. These GZ1 and GZ3 plaques appear sooner than those of KD1 and KD3 (data not shown), no doubt
25 because GZ1 and GZ3 replicate faster because they have a wild-type E1A region.

GZ1 and GZ3 lyse cells and spread from cell to cell much more effectively than *dl*309. At 6 days p.i. of A549 cells, approximately as much monolayer destruction was observed with GZ1 and GZ3 at 10^3 PFU per cell as was observed with *dl*309 at 10^1 PFU per cell (Fig. 15, top panel). This result further underscores the conclusion that overexpression of
30 ADP promotes cell lysis and virus spread.

In theory, GZ1 and GZ3 should be able to replicate not only in tumor cells but also in normal cells. Although they can replicate in normal cells, it is quite possible that GZ1 and GZ3 may be useful as anti-cancer vectors. First, GZ1 and GZ3 could be injected directly into the tumor. Many tumors are self-contained (encapsulated) except for the blood supply. The
35 physical barriers of the tumor could minimize dissemination of the virus to other tissues.

- Second, Ads are in general quite benign. Most infections of Ad5 are in infants and result in mild or asymptomatic disease, and are held in check by strong humoral and cellular immunity. Anti-Ad immunity appears to be life-long. GZ1 and GZ3 could be used only in patients who have an intact immune system, and perhaps also with pre-existing anti-Ad
- 5 immunity. Further, patients could be passively immunized against Ad, using gamma-globulin or even specific purified anti-Ad neutralizing antibodies. Third, considering that Ad5 is a respiratory virus which most efficiently infects lung epithelial cells displaying the specific Ad5 receptor (named CAR) as well as specific integrins (e.g. $\alpha_v\beta_5$), replication-competent vectors derived from Ad5 may not spread efficiently in many non-cancer tissues of the body.
- 10 In addition, it is believed that versions of GZ1 and GZ3 can be constructed that have the E4 promoter substituted with a tumor-specific, tissue-specific, cell-specific, or synthetic promoter. Such vectors would have the positive features associated with wild-type E1A and ADP, and yet be replication-restricted to tumor tissue and/or to particular cell types.

Example 8

15 This example illustrates that the combination of KD1, KD3, GZ1, or GZ3 with radiation is more effective in destroying A549 cells, growing in culture or growing as tumors in nude mice, than the vectors alone or radiation alone.

This was shown in a cell spread assay. A549 cells growing in three 48 well culture dishes were mock-infected or infected with different viruses at multiplicities of infection ranging from 10 to 10^4 PFU per cell as indicated in Fig. 15. One dish was not radiated. A second dish received 600 centrigreys (cGy) of radiation at 24 h p.i., and a third dish received 2000 cGy of radiation at the same time. All dishes were stained with crystal violet at 6 days p.i. With the cells that were not radiated (top panel in Fig. 15), KD1 and KD3 caused monolayer destruction at lower multiplicities of infection than their parental control, d/01/07.

20 25 This was also true for GZ1 and GZ3 as compared to their parental control d/309. (The paucity of cells in the cells infected with GZ1 or GZ3 at 10^4 PFU per cell is an experimental artifact, and is not caused by infection by GZ1 or GZ3). These KD1, KD3, GZ1 and GZ3 results are consistent with earlier results showing that overexpression of ADP leads to increased cell lysis and virus spread.

30 With the dish that was infected then radiated with 600 cGy there was markedly increased cell killing and virus spread as compared to the non-radiated cells (compare the bottom panel of Fig. 15 with the top panel). For example, with KD1, KD3, GZ1, and GZ3 there was about the same amount of cell destruction in the radiated wells at 10^4 PFU per cell as in the non-radiated wells at 10^2 PFU per cell. Similar results were seen with the dish that

received 2000 cGy of radiation (data not shown), and also with dishes that received 600 or 2000 cGy of radiation 24 h prior to infection (data not shown).

The amount of cell destruction was quantitated by extracting the crystal violet from the cells with 33% acetic acid, then measuring the absorbance at 490 nm (data not shown).

- 5 The absorbance with non-radiated mock-infected cells was set at 100% cell viability. With mock-infected cells that received 600 cGy there was a 15% loss in viability (i.e. 15% less crystal violet was extracted). With KD1 at 10^3 PFU per cell, the non-radiated cells were 80% viable whereas the cells receiving 600 cGy of radiation were only about 30% viable. Similar differences in viability between radiated and non-radiated cells were seen with KD3, GZ1,
10 and GZ3. These results argue that the combination of radiation plus vector has a synergistic effect on cell lysis and vector spread, rather than an additive effect. If the effect were only additive, then with the KD1 samples at 10^3 PFU per cell, the cell viability should have been 65% (15% reduction in viability due to radiation alone, 20% reduction due to KD1 alone). In fact, the cell viability was 30% rather than 65%.

- 15 As mentioned, approximately as much cell lysis and virus spread were observed with 600 cGy as with 2000 cGy. To determine the optimal dose of radiation to synergize with the vectors, an experiment similar to the one described above was conducted with mock-, *d/01/07-*, KD1-, KD3-, *d/309*, GZ1-, or GZ3-infected A549 cells. The 48 well plates received 0, 150, 300, or 600 cGy of radiation at 24 h p.i. Cells were stained with crystal violet. The
20 results with cells receiving 0 versus 600 cGy of radiation were similar to those in Fig. 15. The crystal violet was extracted from the cells infected with 10^3 PFU per cell of the difference viruses. The absorbance of crystal violet was determined, and the percent cell viability was graphed, using the absorbance of the non-radiated mock-infected cells as 100% cell viability. As illustrated in Fig. 16, an approximately linear decrease in cell viability in all
25 wells was obtained with increasing radiation dose, although the slope of the line was more negative with KD1, KD3, GZ1, or GZ3 than with mock, *d/01/07*, or *d/309*. With KD1, KD3, GZ1, and GZ3, there was much more cell lysis and vector spread with their parental control viruses, and there was synergy between the vectors and radiation. For example, with mock-infected cells, 600 cGy reduced cell viability by about 30% (70% of cells were viable). KD1 without radiation reduced cell viability by about 23%. The combination of 600 cGy radiation plus KD1 reduced cell viability to about 85%, more than 53% of which is the sum of radiation alone and KD1 alone. When considering the data in Figs. 15 and 16 together, a dose of about 600 cGy is optimal in this type of cell culture experiment.

- 30 The combination of KD3 or GZ3 with radiation was also examined in the A549 tumor-nude mouse model (see Example 4). A549 cells were injected into the hind flanks of
35

nude mice, and tumors were allowed to form. When tumors reached approximately 50- μ l, they were injected with buffer or with 5×10^8 PFU of KD3 or GZ3. Eight to ten tumors were injected per test condition. At 1 day p.i., half the mice received 600 cGy of whole body radiation. Tumor size was measured over time, and was plotted as a fold-increase in tumor size versus days p.i. (as described in Example 4). As shown in Fig. 17, the non-radiated buffer-injected tumors grew faster than those injected with KD3 or GZ3. Tumors that received the combination of KD3 and radiation did not grow, and those that received the combination of GZ3 and radiation shrank in size after 14 days. These results indicate that the combination of KD3 plus radiation or GZ3 plus radiation is more effective than either vector alone or radiation alone in reducing the rate of A549 tumor growth in nude mice. It is likely that radiation would increase the effectiveness in treating tumors of KD1 and GZ1, or indeed any other replication-competent or replication-defective Ad vector.

The mechanism by which radiation causes the ADP overexpressing vectors to lyse cells and spread from cell-to-cell more effectively is not understood. Radiation is expected to induce cellular DNA repair mechanisms, and that may allow for more efficient synthesis of Ad DNA. Radiation may enhance the function of ADP. ADP probably functions by interacting with one or more cellular proteins, and radiation may affect this protein(s) such that ADP functions more efficiently.

It is believed that KD1, KD3, GZ1, or GZ3, or any other replication-competent Ad vector, when used in combination with radiation, will be more effective than vector alone or radiation alone in providing clinical benefit to patients with cancer. The vectors should allow more tumor destruction with a given amount of radiation. Stated another way, radiation should cause more tumor destruction with a given amount of vector. These vectors should also allow the radiation oncologist to use less radiation to achieve the same amount of tumor destruction. Less radiation would reduce the side effects of the radiation.

It is also believed that a cocktail of vectors when used in combination with radiation will be more effective than the cocktail alone or radiation alone. The cocktail could consist of ADP producing vectors plus one or more replication defective vectors expressing an anticancer therapeutic protein (see Example 5).

30

Example 9

This example illustrates a structure-function analysis of adenovirus death protein.

ADP is an 11.6 kDa N-linked O-linked integral membrane glycoprotein that localizes to the inner nuclear membrane (NM) (Scaria et al., Virology 191:743-753). As illustrated in Fig. 18, the Ad2-encoded ADP (SEQ ID NO:6) consists of 101 amino acids; aa 1-40 (SEQ ID NO:17) are luminal, aa 41-59 (SEQ ID NO:18) constitute the transmembrane signal-anchor

(SA) domain, aa 63-70 (SEQ ID NO:19) constitute a basic proline (BP) domain within the nucleoplasmic (NP) domain, which constitutes aa 61-101 (SEQ ID NO:20). To determine which domains in ADP are required to promote cell death, a number of deletion mutants of *rec700* were prepared which lacked various portions of the ADP gene and examined for the 5 ability of ADP to localize to the NM and promote death. The *rec700* virus is an Ad5-Ad-Ad5 recombinant, which has been described elsewhere (Wold et al., *Virology* 148:168-180, 1986).

The structure of ADP in *rec700* and in each deletion mutant is schematically illustrated in Fig. 18. The ADP gene in each deletion mutant has been sequenced using PCR methods to insure that the mutations are correct. The structure and activity of ADP in the 10 deletion mutants was tested by infecting A549 cells followed by immunoblot analysis of the ADP mutant proteins as well as the ability to lyse cells. All deletion mutants expressed a stable ADP protein except *pm734.1* (Δ 1-48, i.e. aa 1-48 are deleted). The *pm734.7* (N_{14}) ADP, which has Asn₁₄ mutated to Ser, is O-glycosylated but not N-glycosylated because Asn₁₄ is the only N-glycosylation site (data not shown). The *dl735* (Δ 4-11) ADP is N- 15 glycosylated but not O-glycosylated because the sites for O-glycosylation are deleted (data not shown). The *pm734.4* (M56) ADP, which has Met₅₆ in the SA domain mutated to Ser, contains exclusively N-linked high-mannose oligosaccharides (data not shown); this occurs because the Met₅₆ mutation precludes exit of ADP from the endoplasmic reticulum (ER). The *dl738* ADP, which lacks aa 46-60 in the signal-anchor domain, forms insoluble aggregates in 20 the cytoplasm; therefore, aa 41-59 do in fact include the signal-anchor domain. The *pm734* (Δ 1-40) ADP, which initiates at Met₄₁ at the N-terminus of the SA domain, comigrated with the lower group of bands generated by proteolytic processing (data not shown). This indicates that the proteolytic cleavage sites occur near Met₄₁. Consistent with this, the 25 proteolytic products were not seen with *dl737* (Δ 29-45) (data not shown). Also, the size of the products decreased in all mutants with deletions within aa 41-101 (*dl715.1*, *dl715*, *dl714*, *dl716*) (data not shown).

The ability of these mutants to promote cell death was monitored by trypan blue exclusion, plaque development, and lactate dehydrogenase release assays (Tollefson et al., *J. Virol.* 70:2296-2306, 1996). The trypan blue results in Fig. 15A indicate that the death-promoting function of ADP was abolished by deletion of aa 1-40 (*pm734*), aa 11-26 30 (*dl736.1*), aa 18-22 (*dl735.1*), or aa 4-11 (*dl735*). Mutation of the N-glycosylation site at Asn₁₄ (*pm734.7*) reduced the death-promoting activity to about 50% of *rec700* (WT). *dl737* (Δ 29-45) was efficient as *rec700* in promoting cell death; this indicates that the proteolytic processing products must not be required to promote cell death because they are not formed 35 with *dl737*. The SA domain is essential for death because *dl738* (Δ 46-60) and *pm734.4*

(M56) were completely defective (Fig. 19). *dl715.1* was nearly completely defective, indicating that the BP domain is extremely important. Surprisingly, aa 71-94 (*dl714*), 76-89 (*dl715*), and 79-101 (*dl716*) could be deleted without affecting the death-promoting activity of ADP (Fig. 19). On the other hand, deletion of aa 81-88 (*dl717*) nearly completely 5 abolished the activity of ADP (Fig. 19); this is probably the result of aberrant sorting of ADP (see below). Similar results were obtained when the ability of these ADP mutants to promote cell death was examined with standard plaque development, LDH-release and MTT assays.

The effects of these mutations on the intracellular localization of ADP are extremely interesting. When examined by immunofluorescence (IF) at 33 h p.i. (data not shown), ADP 10 from *rec700* (WT) localized crisply to the NM; localization to the Golgi was also apparent. With *dl714* (Δ 71-94) and *dl715* (Δ 76-89), ADP localized to all membranes, i.e. the ER, Golgi, plasma membrane, and NM. This was even more apparent at 45 h p.i. (data not shown) Thus, aa 71-94 appear to include a signal that directs ADP specifically to the NM. ADP is very likely sorted from the *trans*-Golgi network (TGN) to the NM, so this putative signal in 15 ADP probably functions in this sorting pathway. ADP from *dl717* (Δ 81-88) is intriguing: it localized to the NM and Golgi, but in many cells "dots" and circular structures were observed. Again, this was more apparent at 45 h p.i. when these structures were the prominent feature. *dl717*-infected cells have not begun to die at 45 h p.i., so these structures are not cellular remnants. The intriguing possibility is that these structures are membrane vesicles that have 20 pinched off from the TGN but are defective in targeting to and/or fusing with the NM.

With *dl738* (Δ 46-60 in the SA domain), ADP aggregated in the cytoplasm. This again indicates that aa 46-60 include the SA sequence. With *pm734.4* (M56), ADP localized primarily to the NM. As discussed above, the *pm734.4* ADP has exclusively high-mannose N-linked oligosaccharides, indicating that it never leaves the ER. Perhaps the putative NM-localization signal in the C-terminal region of the *pm734.4* ADP targets ADP to the NM by 25 lateral diffusion from the ER (which is continuous with the outer and inner NM).

With *dl737* (Δ 29-45), ADP localized to the NM. ADP from *pm734* (Δ 1-40), *pm734.7* (N14) (N-linked glycosylation cannot occur), and *dl735* (Δ 4-11; the O-glycosylation sites are deleted) localized much more prominently to the Golgi than the NM. ADP from *dl735.1* (Δ 18-22) and *dl736.1* (Δ 11-26) also localized much more strongly to the Golgi than the NM. Thus, residues 1-26 and/or glycosylation appear to be required for efficient transport of ADP 30 from the Golgi/TGN to the NM.

In summary, aa 41-59 include the SA domain, Met₅₆ in the SA domain is required for exit from the ER, aa 1-26 are required for efficient exit from the Golgi, and aa 76-94 are 35 required to target ADP specifically to the NM. With respect to promoting cell death, the

essential regions are aa 1-26, the SA domain (ADP does not enter membranes), Met₅₆ in the SA domain, and the BP domain (aa 63-70). It is not clear whether the defective death-promoting phenotype of *pm734* (Δ 1-40), *dl735* (Δ 4-11), *dl735.1* (Δ 18-22), *dl736.1* (Δ 11-26), and *pm734.7* (N14) is due to lack of sequences (or oligosaccharides) that promote death or to much slower exit of ADP from the Golgi to the NM. *dl714* (Δ 71-94) and *dl715* (Δ 76-89) express a wild-type phenotype for promoting death even though they are defective in localizing specifically to the NM; this is probably because sufficient ADP still enters the NM to promote death. Even though the deletion in *dl717* (Δ 81-88) lies within the deletions in *dl715* (Δ 76-89) and *dl714* (Δ 71-94), the *dl717* ADP is only about 15% as efficient as *rec700* (WT), *dl715* and *dl714* in promoting death. This may be because the *dl717* ADP tends to remain in vesicles rather than localizing to the NM. Altogether, these data indicate that ADP must localize to the NM in order to promote cell death.

Example 10

This example further characterizes the tissue specific Ad vectors described in Example 6. As discussed therein, the Ad E4 promoter is deleted and replaced with the promoter for surfactant protein B (SPB) in these vectors (Figure 24).

Materials and Methods

Cells, vectors and methods described in Example 6 were also used in this Example. In addition to the human cancer cell lines A549 (human lung carcinoma), Hep 3B (human hepatocellular carcinoma), and H441 (papillary lung adenocarcinoma) used in Example 6, HEK 293 cells (obtained from Microbix (Toronto, ON)) and VK10-9 cells were used. VK10-9 cells are 293 cells that in addition to E1 contain and express E4 and pIX. These cells will be referred to as 293-E4 cells.

Experiments employing phase contrast microscopy of Hep 3B and H441 cells were performed as follows. Monolayers of Hep 3B or H441 cells were grown in 60 mm dishes with 5 ml of DMEM (10% FBS), and were mock-infected or infected with KD1 or KD1-SPB at a multiplicity of infection of 10 plaque forming units (PFU) per cell. Phase contrast photographs of monolayers were taken at 4 and 7 days postinfection (p.i.).

Experiments employing western blots of H441 or Hep 3B cells were performed as follows. H441 or Hep 3B cells (in 60 mm dishes) were infected with 10 PFU/cell of KD1 or KD1-SPB. At 24 h p.i., the cells were washed three times with PBS and harvested by scraping. The cells were lysed by RIPA buffer. The protein concentration was measured by the BIO-RAD DC Protein Assay Kit (BIO-RAD Laboratories, Hercules, CA) and 10 μ g of each sample were electrophoresed on 15% sodium dodecylsulfate polyacrylamide gels (SDS-PAGE). The gels were electroblotted onto PVDF membranes (Immobilon, Millipore,

Bedford, MA). The membranes were blocked in TBST (50 mM Tris-Cl, pH 7.6, 150 mM NaCl, 0.2% Tween 20) containing 10% dry milk (Carnation) overnight at 4°C. After blocking, the membranes were incubated with a rabbit polyclonal antiserum against E4ORF3 (gift of Gary Ketner) or ADP (Tollefson et al., *J. Virol.* 66:3633-3642, 1992), or with M73, a 5 monoclonal antibody against E1A (Harlow et al., *J. Virol.* 55:533-546, 1985). The secondary antibodies were goat anti-rabbit IgG-HRP or goat anti-mouse IgG-HRP. The blots were developed using the ECL protocol (Amersham Pharmacia, Arlington Heights, IL).

Experiments employing a lactate dehydrogenase release assay for cell lysis (Tollefson et al., *J. Virol.* 70:2296-2306) were performed as follows. H441 cells (7.7×10^5 cells per 35 10 mm dish) and Hep 3B cells (9.0×10^5 cells per 35 mm dish) were infected at 20 PFU/cell in one ml serum-free DMEM. After an adsorption period of 1 h, 3 ml of DMEM (10% FBS) were added (final FBS concentration of 7.5%). Cells were incubated at 37°C with 6% CO₂. At daily intervals, supernatants were collected, microfuged to remove floating cells, and cell-free supernatants were frozen at -70°C until assayed. Total lysis samples were prepared by 15 addition of 10X lysis buffer included in the Cyto Tox 96 kit (Promega, Madison, WI). After all samples were collected, 20 µl samples were assayed in triplicate using the LDH assay kit Cyto Tox 96 and read on an EL340 Microplate reader (BioTecTM Instruments, Inc.) at 490 nm.

Experiments employing immunofluorescence evaluation of H441 and Hep 3B cells 20 were performed as follows. H441 and Hep 3B cells were plated on Corning #1 coverslips in 35 mm dishes. H441 (1.5×10^6 cells/35 mm dish) and Hep 3B (9.0×10^5 cells/35 mm dish) were infected with 20 PFU/cell of the indicated viruses in 1 ml serum-free DMEM. After 1 h, 1 ml of DMEM/20% FBS was added (final concentration of 10% FBS). At the indicated times (48 h or 6 d p.i.), cells were fixed for 10 min in 3.7% paraformaldehyde in PBS, then 25 permeabilized for 6 min in methanol (-20°C) and rehydrated in PBS. Coverslips were stained with rabbit antipeptide antiserum against the Ad E2A-coded DNA binding protein (DBP) (1:400 dilution; gift of Maurice Green) and mouse monoclonal antibody against fiber (1:400 dilution; gift of Jeff Engler) or were stained with rabbit antiserum to E4ORF3 (1:250 dilution; gift of Gary Ketner). Secondary antibodies (Cappel/ICN) were used at 1:50 dilution. All 30 antibodies were diluted in PBS containing 1% BSA and 0.1% sodium azide. Photographs were taken on a Nikon epifluorescence microscope using a 100X Planapo lens and Tmax 400 film (Kodak). The film was developed in Diafine developer.

Analysis of viral DNA replication by Southern hybridization was performed as follows. H441 and Hep 3B cells were grown in 60 mm dishes in DMEM supplemented with 35 10% FBS. Cells were infected at 70% confluence with 10 PFU/cell of KD1 or KD1-SPB.

Dishes were incubated in humidified 5% CO₂ atmosphere at 37°C. Total genomic DNAs were isolated at 5, 24, 48, 72, and 96 h p.i. Equal amounts of total genomic DNAs were digested with HindIII and resolved on a 1% agarose gel prior to transfer onto membranes. A random primer ³²P-labeled pBHG10 plasmid probe (Bett et al., *Proc. Natl. Acad. Sci. USA* 91:8802-8806, 1994) was used for hybridization, and the blots were autoradiographed. DNA fragments were quantitated on a Molecular Dynamics PhosphorImager.

Virus yields were determined as follows. Hep 3B cells or H441 cells grown as monolayers in 35 mm dishes were infected with 10 PFU/cell of KD1 or KD1-SPB. At days 0 to 4 (for H441) or days 0 to 9 (for Hep 3B) p.i., cells and culture medium were frozen at -70°C. Samples were frozen and thawed three times to release the virus from the cells, and total virus yields were determined by plaque assay on A549 monolayers.

The effect of KD1-SPB and KD1 on H441 and Hep 3B tumors was examined in a nude mouse model (Doronin et al., *J. Virol.* 74:6147-6155, 2000). Tumor cells (10⁷ cells in 200 µl of DMEM, 50% Matrigel [Becton Dickinson Labware, Bedford, MA] for H441 cells, or 10⁷ cells in 200 µl of DMEM plus 10% Matrigel for Hep 3B cells) were injected into flanks of 5-6 weeks old athymic nude mice and allowed to grow for three weeks to about 100 µl (H441) or 150 µl (Hep 3B) volumes. Pre-established tumors (n = 10) were injected with 50 µl of DMEM or 5 x 10⁷ PFU of indicated viruses in DMEM. Injections of the viruses were repeated twice weekly for 3 weeks to the total dose of 3.0 x 10⁸ PFU per tumor. Tumor size measurements were taken twice per week for H441 cells, or weekly for Hep 3B cells using a Sylvac digital caliper. Tumor volumes were calculated in according to the formula: length x width²/2. Data are represented as means of increase in tumor size relative to the tumor size at the initial injection.

Results

The properties of KD1-SPB in various cell types were compared to those of its "parent", KD1. Figure 25 shows the plaque development properties of these vectors on 293-E4, 293, and A549 cells. The data are plotted as the number of plaques seen on any day of the plaque assay as a percentage of the number of plaques seen at the end of the assay (i.e. when new plaques cease to appear) (Tollefson et al., *J. Virol.* 70:2296-2306, 1966). This assay is an indicator of the size of the plaques. KD1 formed plaques equally well on 293-E4 and 293 cells (Figure 25A). With KD1-SPB, plaques were observed about 3-4 days sooner on 293-E4 compared to 293 cells (Fig. 2A). On A549 cells, KD1 formed plaques 4-6 days sooner than KD1-SPB (Figure 25B).

The properties of KD1-SPB versus KD1 were characterized in detail in H441 cells, a human papillary lung adenocarcinoma cell line known to express the TTF1 transcription

4.5

factor and in which the SPB promoter is active (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). Hep 3B cells, a human hepatocellular carcinoma in which the SPB promoter should not be active, were used as a negative control. H441 and Hep 3B monolayers were infected with 10 PFU/cell of KD1 or KD1-SPB and photographed at 4 and 7 days p.i. Mock-infected 5 Hep 3B cells formed a relatively homogeneous monolayer, but H441 cells tended to form structures that resemble syncytia (Figure 26A, B). As expected, KD1 produced cytopathic effect (CPE) on both cell lines at 4 and 7 days p.i. (Figure 26A, B). Also as expected, KD1-SPB caused CPE on H441 cells but not on Hep 3B cells. Since CPE in Ad-infected cells is usually an indicator of virus growth, these results suggest that KD1-SPB grows in H441 but 10 not in Hep 3B cells.

To examine viral DNA replication, H441 and Hep 3B cells were infected with 10 PFU/cell of KD1 or KD1-SPB, then the accumulation of viral DNA was determined by DNA blot. With H441 cells, KD1 and KD1-SPB DNAs were readily detected at similar levels at 48-96 h p.i. (Figure 27A). With Hep 3B cells, KD1 DNA levels were similar to those in 15 H441 cells, but KD1-SPB DNA was barely detectable. This was confirmed by PhosphorImager analysis of the DNA bands (Figure 27B).

Growth of KD1-SPB and KD1 in H441 and Hep 3B cells was determined by a single step growth assay. Cells were infected with 10 PFU/cell of vector, then total vector yield was determined by plaque assay. Total yield of both vectors was similar in H441 cells, reaching a 20 plateau after 2 days (Fig. 28A). KD1 yield plateaued in Hep 3B cells after 2-4 days p.i. (Figure 28B). However, KD1-SPB levels were about 5 logs lower in Hep 3B cells after 2-4 days, and even by 9 days they had not achieved the levels of KD1. We conclude that KD1-SPB grows with significant specificity on H441 versus Hep 3B cells. Further, KD1-SPB grows as well as KD1 on H441 cells, indicating that the E4 promoter deletion by itself does 25 not significantly compromise the vector, and that the E4 promoter can be replaced by a tissue-specific promoter in a replication-competent vector.

To obtain further details on the replication of KD1-SPB vs KD1 in H441 and Hep 3B cells, the expression of representative Ad proteins by KD1-SPB and KD1 was examined. H441 or Hep 3B cells were mock-infected or infected with 10 PFU/ml of KD1 or KD1-SPB, 30 then at 24 h p.i. the proteins were extracted and the E1A, E4ORF3, and ADP proteins were examined by immunoblot. E4ORF3 is one of the six proteins coded by the E4 transcription unit (Leppard, *J. Gen. Virol.* 78:2131-2138, 1997). As anticipated, KD1-SPB expressed E4ORF3 well in H441 cells, but only at trace levels in Hep 3B cells (Figure 29). KD1-SPB expressed the E1A proteins in Hep 3B cells. Synthesis of E1A proteins by KD1-SPB in Hep 35 3B cells is expected because E1A expression does not require E4 proteins; it also indicates

that the block to infection with KD1-SPB is downstream of E1A. KD1 expressed E1A in both cell lines, but the amount was less than obtained with KD1-SPB in Hep 3B cells (Figure 29). The increased E1A levels seen with KD1-SPB may reflect its poor ability to enter the late phase of infection (see Discussion). KD1-SPB expressed ADP as well as KD1 in H441 cells, but it did not make detectable ADP in Hep 3B cells. ADP is primarily a late protein, so this result is consistent with the relative lack of E4 protein expression, DNA replication, and growth of KD1-SPB in Hep 3B cells.

To gain insights into replication events that occur in individual cells, expression of E4ORF3, the E2A-DBP, and the fiber late protein was examined by immunofluorescence. 10 H441 or Hep 3B cells were infected with 20 PFU/cell. At 48 h or 6 days p.i., cells were fixed and immunostained. E4ORF3 was detected in the nuclei of H441 cells at 48 h p.i. with KD1, KD1-SPB, or dl309 (Figure 30A). (dl309 is an Ad5 mutant that has wild-type E1A, expresses Ad5 levels of ADP, and lacks the E3-RID and E3-14.7K genes). E4ORF3 could not be detected in the vast majority of Hep 3B cells infected with KD1-SPB (Figure 30A), even at 6 15 days p.i. (Figure 30B). Thus, KD1-SPB expresses E4ORF3 well in H441 but not in Hep 3B cells.

Figure 31A shows double label immunofluorescence of DBP and fiber in the same Hep 3B cells at 48 h p.i. with KD1 or KD1-SPB. With KD1, there was a strong speckled staining pattern in the nucleus that is typical for DBP at 48 h p.i. (Figure 31A, top left panel). 20 There was strong staining of fiber throughout these same cells (Figure 31A, top right panel). Staining of the cytoplasm and nucleus is expected because fiber is synthesized in the cytoplasm and then transported to the nucleus where virions assemble. With KD1-SPB at 48 h p.i., about 25% of the cells showed the speckled staining for DBP, and only one cell (7% of total) with the advanced speckled pattern was also stained for fiber (Figure 31A, bottom two 25 panels). Even at 6 days p.i., only about 30% of cells showed staining for DBP, and about 20% for fiber (Figure 31B). Thus, markedly fewer Hep 3B cells infected with KD1-SPB expressed DBP and especially fiber as compared to KD1. These results indicate that KD1-SPB replicates as well as KD1 in H441 cells, no doubt because the SPB promoter is active in H441 cells (Yan et al., *J. Biol. Chem.* 270:24852-24857, 1995). KD1-SPB barely replicates 30 in Hep 3B cells, presumably because the SPB promoter is minimally active in these cells.

At the culmination of replication, Ad-infected cells are lysed and the virus spreads to other cells; this process is mediated in large part by ADP (Tollefson et al., *Virology* 220:152-162, 1996; Tollefson et al., *J. Virol.* 70:2296-2306, 1996). To examine vector-induced cell lysis, H441 and Hep 3B cells were mock-infected or infected with 20 PFU/cell of KD1, KD1-SPB, or dl309, and cell lysis was determined by release of lactate dehydrogenase (Tollefson et 35

al., *J. Virol.* 70:2296-2306, 1996). All vectors lysed H441 cells beginning at 2-3 days p.i. (Figure 32A). KD1 and dl309 also lysed Hep 3B cells in the same time period; however, KD1-SPB caused only minimal cell lysis (Figure 9B). Thus, these data, along with the cell spread data in Example 6 and Figure 13, demonstrate that KD1-SPB lyses cells and spreads 5 efficiently from cell-to-cell in H441 but not Hep 3B cells.

An experiment was conducted to determine whether KD1-SPB or KD1 would suppress H441 tumors in nude mice. H441 cells were injected into each hind flank. When tumors had grown to about 100 µl (H441) or 150 µl (Hep 3B), they were injected twice weekly for 3 weeks with DMEM (mock) or 5×10^7 PFU of test virus in 50 µl of DMEM (3.0 10 $\times 10^8$ total PFU). Ten tumors (5 mice) were used for each virus. Growth of H441 tumors was suppressed similarly by KD1-SPB and KD1 (Figure 33A). KD1 suppressed growth of Hep 3B tumors, whereas KD1-SPB caused only minimal suppression (Figure 33B). These results show that KD1-SPB is as effective as KD1 in suppressing tumors when the SPB promoter is active. Further, the cell type specificity observed with KD1-SPB in vitro is maintained in 15 vivo.

Discussion

Tumor specificity is one of the biggest challenges facing cancer gene therapy, i.e. having the therapeutic gene be expressed specifically in cancer cells. Specificity is very important for RC viruses. Two main strategies have been described that in theory confer 20 specificity: transductional targeting and transcriptional targeting. Directing specificity of vectors toward specific cell surface receptors on the target cells has been attempted through various methods. Although this approach is theoretically attractive it might encounter multiple obstacles such as the lack of incorporation of the engineered protein into the virion (Scaria et al., *Virology* 191:743-753, 1992) or lack of infectivity through the targeted receptor 25 (Cosset et al., *J. Virol.* 69:6314-6322, 1995). Transcriptional targeting utilizes tumor and tissue specific promoters. In replication-defective vectors these regulatory sequences confine the expression of cytotoxic genes to specific tissues. In replication-competent vectors, as an added layer of regulation, vector replication per se can be placed under the control of tumor or tissue specific promoter/enhancer sequences. In replication-competent Ad, insertion of the 30 tissue or tumor specific promoter/enhancer into the E1A promoter/enhancer region has been used exclusively (Hallenbeck et al., *Hum. Gene Ther.* 10:1721-1733, 1999; Rodriguez et al., *Cancer Res.* 57:2559-2563, 1997; Yu et al., *Cancer Res.* 59, 4200-4203, 1999; Yu et al., *Cancer Res.* 59:1498-1504, 1999). The rationale behind these vectors is that expression of E1A and therefore the whole Ad transcription program will depend on these tissue or tumor 35 specific promoters. However, as a generic approach, there may be difficulties. The E1A

enhancer/promoter is very complex. The enhancer controls not only the E1A promoter but also distant promoters such as the E4 promoter (Shenk, T. pp. 2111-2148 *In* B.N. Fields, D.M. Knipe, and P.M. Howley (eds.), *Fields Virology*, Lippincott-Raven, Philadelphia, 1996). In addition, it has been shown that the E1A enhancer in the inverted terminal repeat 5 region changes tissue specificity of cellular promoters (Shi et al., *Hum. Gene Ther.* 8:403-410, 1997). Also, the E1A enhancer/promoter is partially embedded within the signals required to package the Ad genome into virions, and it may be problematic to remove all the E1A enhancer elements without impairing virus production. Accordingly, we chose to replace the E4 promoter with a tissue specific promoter. E4 genes are essential for Ad 10 replication, and therefore we expected that the replication of the recombinant virus would be dependent on the tissue specific regulatory elements.

To construct KD1-SPB, the ca. 300 bp of the E4 promoter was deleted and the B-500 version (ca. 500 bp) of SPB promoter was inserted (Yan et al., *supra*) (Figure 24 C, D). We selected the SPB promoter because of its strict tissue specificity: it is exclusively active in 15 type II alveolar cells and bronchial epithelial cells of the lung (Bohinski et al., 1994, *Mol. Cell. Biol.* 14:5671-5681, 1994). Since the parental virus KD1 contains and expresses two E1A mutations that restrict virus replication to tumor cells (Doronin et al., *supra*), we anticipated that the virus would selectively replicate in cells derived from lung tumors. Thus, H441 cells, a papillary lung carcinoma cell line, were used to characterize the replication, 20 gene expression, and functional profile of KD1-SPB.

KD1-SPB formed plaques 3-4 days sooner on 293-E4 cells that express E4 proteins than on 293 cells, whereas KD1 formed plaques with the same kinetics on both cell lines. These data show that the E4 promoter is active in 293 cells, and that the SPB promoter displays very low activity in 293 cells. It is not clear why KD1-SPB forms plaques on 293 25 cells; these cells are derived from human embryonic kidney and at least one of the transcription factors regulating the SPB promoter (Bohinski et al., *supra*), hepatocyte nuclear factor 3, is expressed in embryonic kidney. It is also possible that TTF1, the master regulatory factor of SPB expression, is minimally active in 293 cells.

KD1 grew to equally high titers in H441 and Hep 3B cells (Figure 28A, B). In 30 contrast, KD1-SPB replicated as efficiently as KD1 in H441 cells, in which the SPB promoter is active (Yan et al., *supra*) (Figure 28A), but replicated poorly in Hep 3B cells, most likely because the SPB promoter is inactive (Figure 28B). This selectivity has been confirmed by measuring viral DNA production in the two cell lines. KD1-SPB DNA replication was similar both kinetically and quantitatively to KD1 DNA replication in H441, however in Hep

3B cells, KD1-SPB DNA was almost undetectable (Figure 27A, B). The cytopathic effect, a surrogate marker of Ad replication, showed a similar specificity (Figure 26).

To further confirm our predictions on the molecular basis of the observed issue specificity we monitored viral protein expression. When cells were infected with KD1-SPB
5 all the viral proteins early or late, except for E1A, were expressed in a tissue-specific fashion (high expression in H441, low to undetectable expression in Hep 3B) (Figures 29-31). We found a good correlation between the levels of E4 promoter activity (E4ORF3 expression) and the expression of E2A-DBP, ADP, and fiber proteins. Thus, the SPB promoter retains its tissue specificity in the Ad genome and it seems to be the limiting factor of Ad gene
10 expression in the cell lines tested. As expected, expression of E1A is not tissue-specific. Thus, the regulatory step of tissue-specific Ad DNA replication is downstream of E1A. In Hep 3B cells, KD1-SPB expressed E1A at a higher level than did KD1 (Figure 29), strongly suggesting that KD1-SPB replication in most of the Hep3B cells remains at the early stage.

The cytolytic effect of KD1-SPB also showed a tissue-specific profile (Figure 32; Figure 13 of Example 6), i.e., preferential lysis of H441 cells over Hep 3B cells, a pattern similar to the specificity observed at the level of DNA replication (Figure 27) and viral protein synthesis (Figures 29-31). This cell type specificity was also observed when these cells were growing as tumors in nude mice. Growth of H441 tumors was suppressed by KD1-SPB and KD1 at similar efficacy (Figure 33A). In contrast, KD1-SPB unlike KD1 had only
20 minimal effect on the growth of Hep 3B tumors (Figure 33B).

In summary, substitution of the E4 promoter with a tissue specific promoter allows highly tissue specific replication of Ad vectors and in the target tissue it is as efficient as the replication of the parental virus. KD1-SPB lacks all E3 genes except ADP. E3 gp19K, RID and 14.7K have been shown to protect Ad-infected cells from attack by cytotoxic
25 lymphocytes and apoptosis-inducing cytokines such as tumor necrosis factor and Fas ligand (Wold et al., pp. 200-232 *In A.J. Cann (ed.), DNA Virus Replication: Frontiers in Molecular Biology*, Oxford University Press, Oxford, 2000; Wold et al., *Curr. Opin. Immunol.* 11:380-386, 1999).

The therapeutic index (virus produced in H441 cells compared to Hep 3B cells) of
30 KD1-SPB is 10^4 - 10^5 for the first 4-5 days (Figure 28). These data compare to data reported by Calydon (10^4 - 10^5) for their prostate specific viruses (Rodriguez et al., *supra*; Yu et al., *Cancer Res.* 59, 4200-4203, 1999; Yu et al., *Cancer Res.* 59:1498-1504, 1999). We suggest that KD1-SPB has some added advantage over vectors reported by other laboratories because it encodes a mutant form of E1A that restricts replication to cancer cells (Doronin et al.,
35 *supra*).

Although the lung ranks as the second highest cancer site for both men and women in the U.S. Reis et al., *Cancer Res.* 88:2398-2424, 2000), lung cancer has not been a major target for cancer vector gene therapy since intratumoral injection of virus is generally not feasible in the lungs. However, there has been a recent report of intratumor injection of a replication-defective Ad vector into a lung tumor, and such an approach could be attempted with KD1-SPB. It may also be feasible to administer KD1-SPB systemically in the lung.

In view of the above, it will be seen that the several advantages of the invention are achieved and other advantageous results attained.

As various changes could be made in the above methods and compositions
10 without departing from the scope of the invention, it is intended that all matter contained in the above description and shown in the accompanying drawings shall be interpreted as illustrative and not in a limiting sense.

All references cited in this specification, including patents and patent applications, are hereby incorporated by reference. The discussion of references herein is
15 intended merely to summarize the assertions made by their authors and no admission is made that any reference constitutes prior art. Applicants reserve the right to challenge the accuracy and pertinence of the cited references.

What is Claimed Is:

1. A recombinant vector which is replication-competent in a neoplastic cell and which overexpresses an adenovirus death protein.
2. The recombinant vector of claim 1 wherein the adenovirus death protein comprises amino acids 1-26, 41-59, and 63-70 of SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8 or a conservatively substituted variant thereof or wherein the adenovirus death protein comprises SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8.
3. The recombinant vector of claim 2 which comprises a recombinant virus.
4. The recombinant vector of claim 3, wherein the recombinant virus is an adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.
5. The recombinant vector of claim 4 which comprises SEQ ID NO:3 or SEQ ID NO:4.
6. The recombinant vector of claim 3 which is replication-restricted to neoplastic cells.
7. The recombinant vector of claim 6 which comprises SEQ ID NO:1 or SEQ ID NO:2.
8. The recombinant vector of claim 3, wherein the recombinant adenovirus comprises a tissue specific promoter, a tumor specific promoter, or an inducible promoter substituted for the E4 promoter.
9. The recombinant vector of claim 8, wherein the tissue-specific promoter is a surfactant protein B promoter.
10. The recombinant vector of claim 6 which comprises SEQ ID NO:14, SEQ ID NO:15 or SEQ ID NO:16.
11. The recombinant vector of claim 1, wherein the vector further comprises a gene encoding an anti-cancer product.
12. The recombinant vector of claim 11, wherein the gene encoding an anti-cancer product is in the E3 region of the vector.
13. A method for promoting death of a neoplastic cell comprising contacting the neoplastic cell with at least one vector which is replication competent in the neoplastic cell and which overexpresses an adenovirus death protein.
14. The method of claim 13 wherein the adenovirus death protein comprises amino acids 1-26, 41-59, and 63-70 of SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ

ID NO:8 or a conservatively substituted variant thereof or wherein the adenovirus death protein comprises SEQ ID NO:5, SEQ ID NO:6, SEQ ID NO:7, or SEQ ID NO:8.

15. The method of claim 14, wherein the vector comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.

16. The method of claim 15, wherein the neoplastic cell comprises a tumor in a patient and the contacting step comprises administering the recombinant adenovirus to the tumor.

17. The method of claim 16, further comprising the step of passively immunizing the patient against the recombinant adenovirus.

18. The method of claim 17, wherein the recombinant adenovirus comprises SEQ ID NO:3 or SEQ ID NO:4.

19. The method of claim 15, wherein the vector is replication-restricted to neoplastic cells.

20. The method of claim 19, wherein the vector is a recombinant adenovirus comprising SEQ ID NO:1 or SEQ ID NO:2.

21. The method of claim 15, wherein the recombinant adenovirus comprises a tissue specific promoter or an inducible promoter substituted for the E4 promoter.

22. The method of claim 21, wherein the tissue specific promoter is a surfactant protein B promoter.

23. The method of claim 22, wherein the recombinant adenovirus comprises SEQ ID NO:14, SEQ ID NO:15 or SEQ ID NO:16.

24. The method of claim 16, further comprising treating the tumor with radiation.

25. The method of claim 24, comprising administering more than one recombinant adenovirus to the tumor and treating the tumor with radiation.

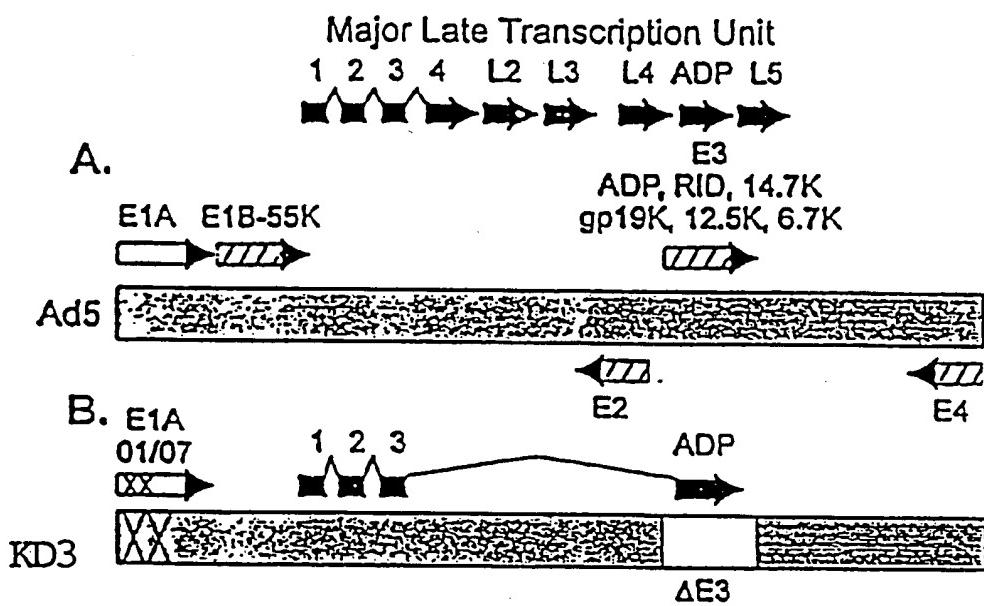
26. The method of claim 16, further comprising treating the tumor with chemotherapy.

27. The method of claim 26, comprising administering more than one recombinant adenovirus to the tumor and treating the tumor with chemotherapy.

28. The method of claim 16, further comprising administering to the tumor one or more replication-defective adenovirus which expresses an anti-cancer gene product, wherein the recombinant adenovirus complements spread of the replication-defective adenovirus in the tumor.

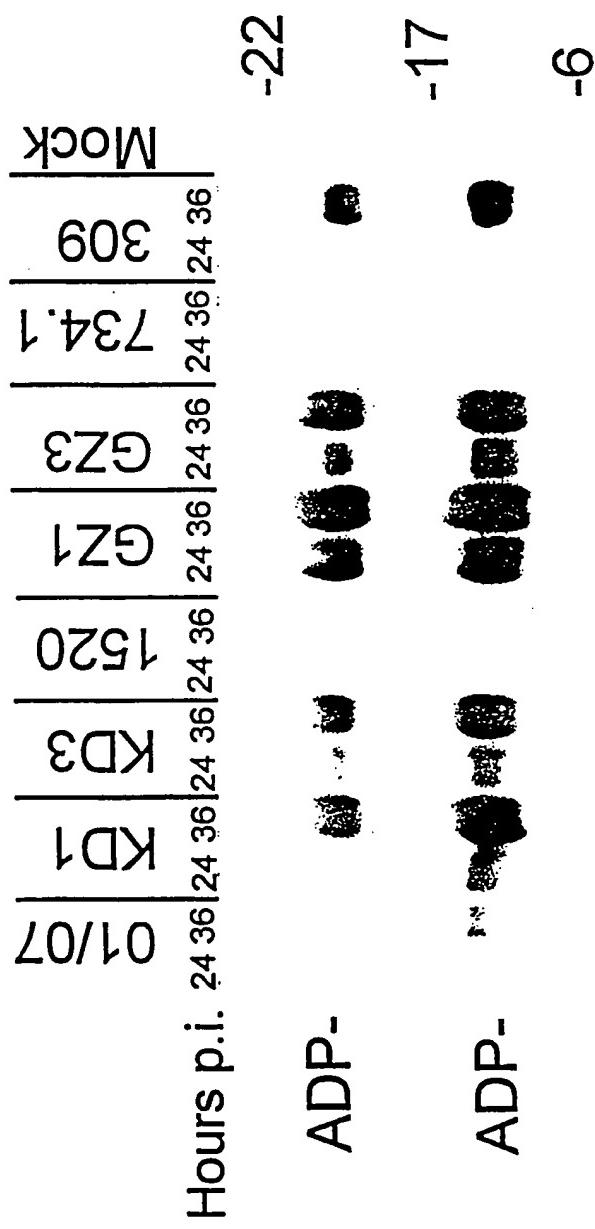
29. A composition comprising:

- a first recombinant virus which is replication competent in a neoplastic cell and overexpresses an adenovirus death protein; and
- 5 a second recombinant virus which is replication defective and which expresses an anti-cancer gene product,
- wherein the first recombinant virus complements replication of the second recombinant virus.
30. The composition of claim 29 wherein the first recombinant virus comprises a recombinant adenovirus lacking expression of at least one E3 protein selected from the group consisting of: gp19K; RID α ; RID β and 14.7K.
31. The composition of claim 30 wherein the recombinant adenovirus comprises a nucleotide sequence selected from the group consisting of: SEQ ID NO:1; SEQ ID NO:2; SEQ ID NO:14; SEQ ID NO:15; SEQ ID NO:16; SEQ ID NO:3; or SEQ ID NO:4.
32. A composition comprising
- a first recombinant virus which is replication-defective in a neoplastic cell and which overexpresses an adenovirus death protein, and
- a second recombinant virus which is replication-competent in a neoplastic cell.

**FIGURE 1**

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ADP Is Expressed Earlier in Infection
By KD1, KD3, GZ1, and GZ3



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FIGURE 2

**The E1A 01/07 Mutation
Retards Late Gene Expression**

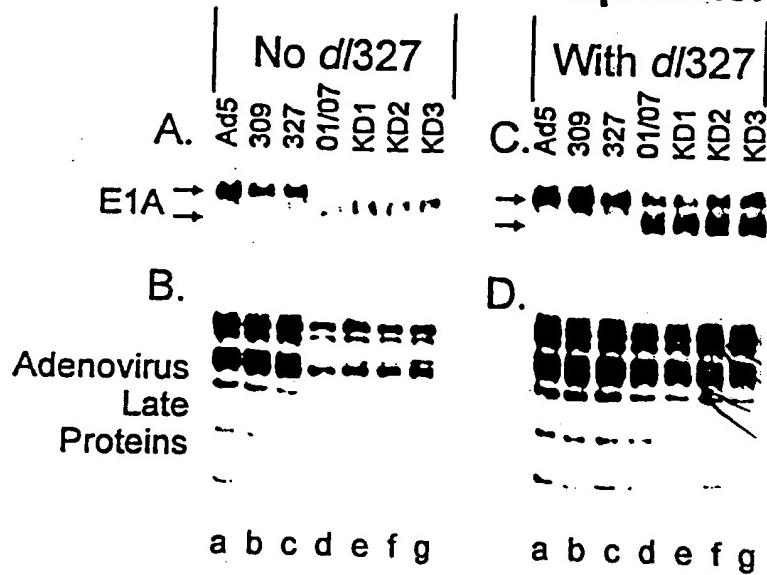


FIGURE 3

3/66

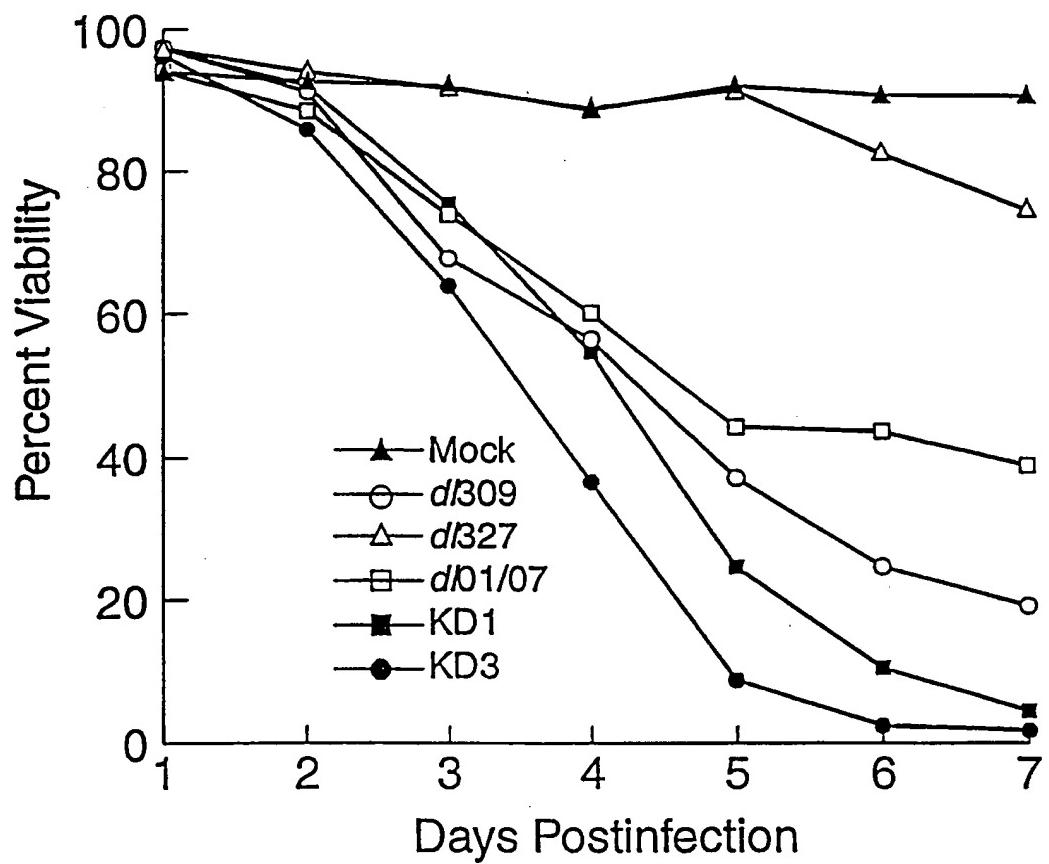


FIGURE 4

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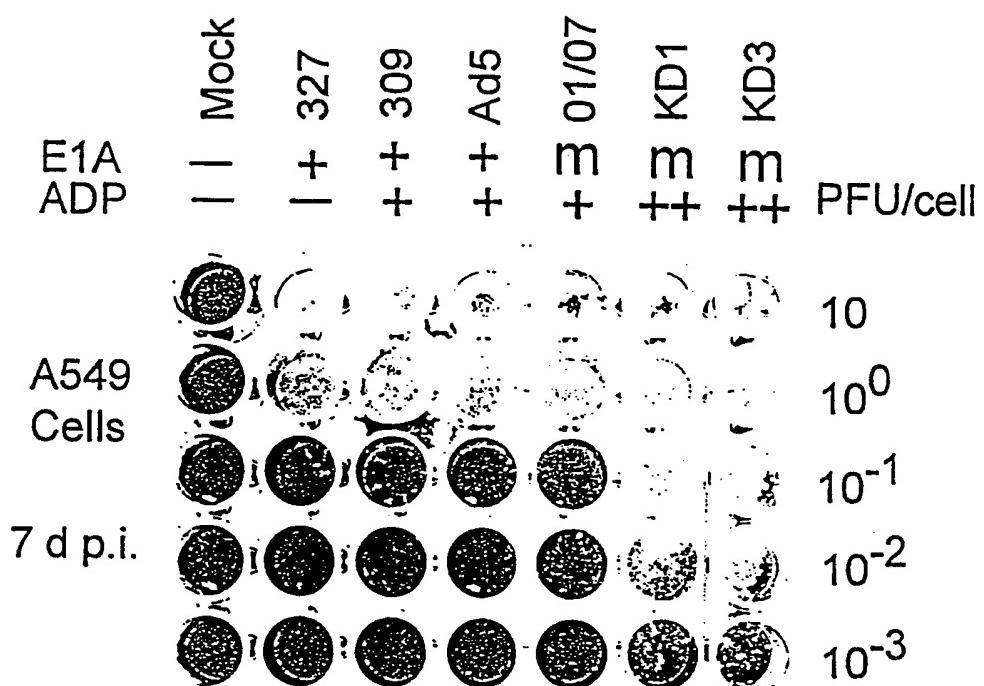


FIGURE 5

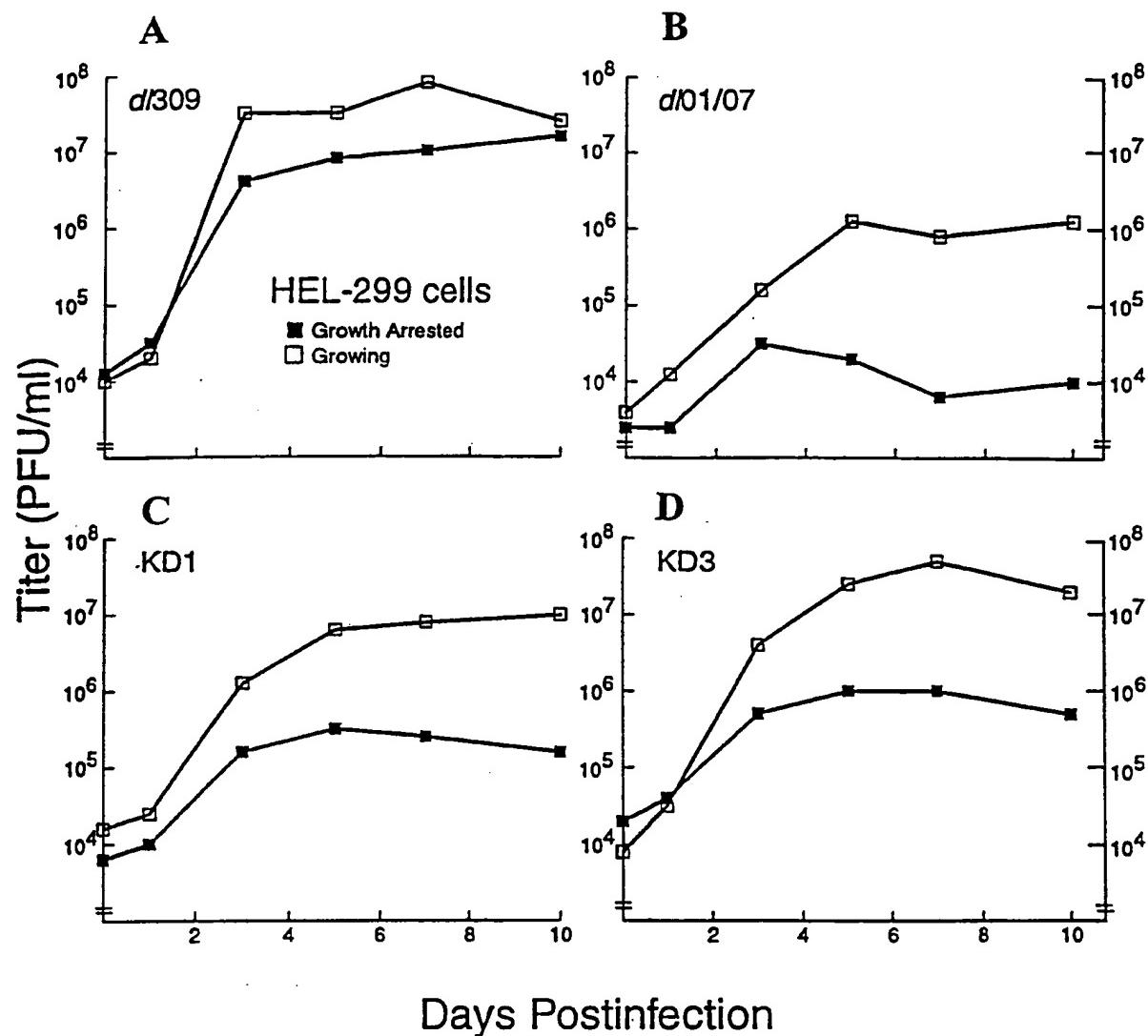


FIGURE 6

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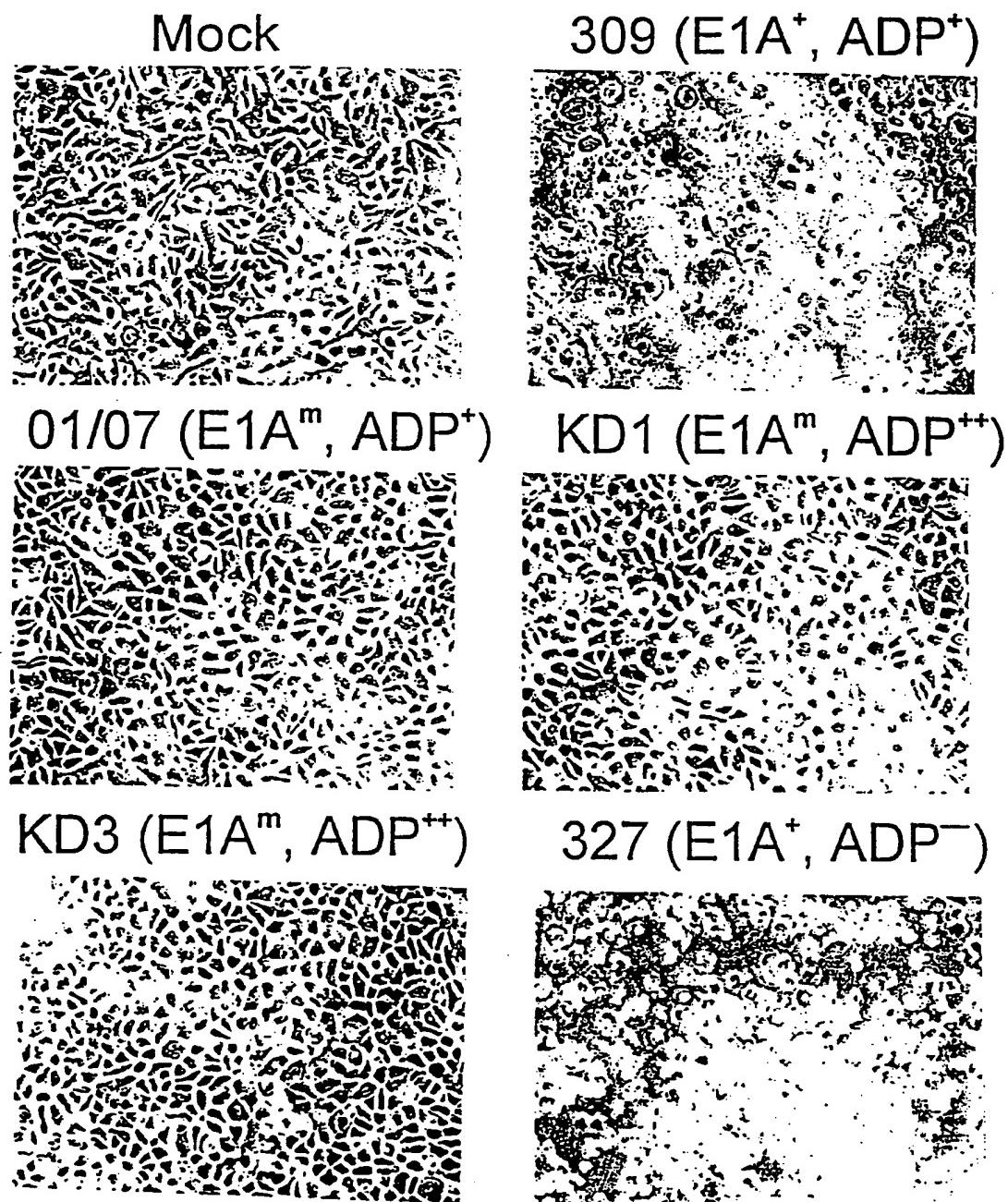


FIGURE 7

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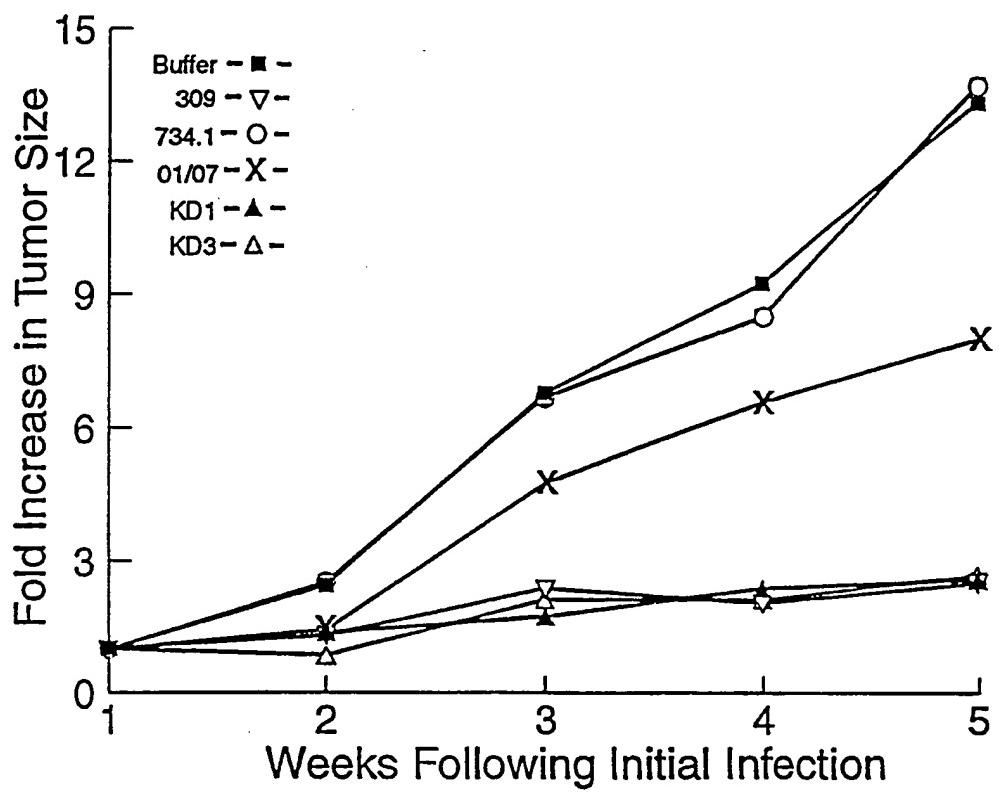


FIGURE 8A

8/14/04

**One Injection of KD3 or GZ3 Inhibits
Growth of A549 tumors
(5×10^8 PFU injected on day 0)**

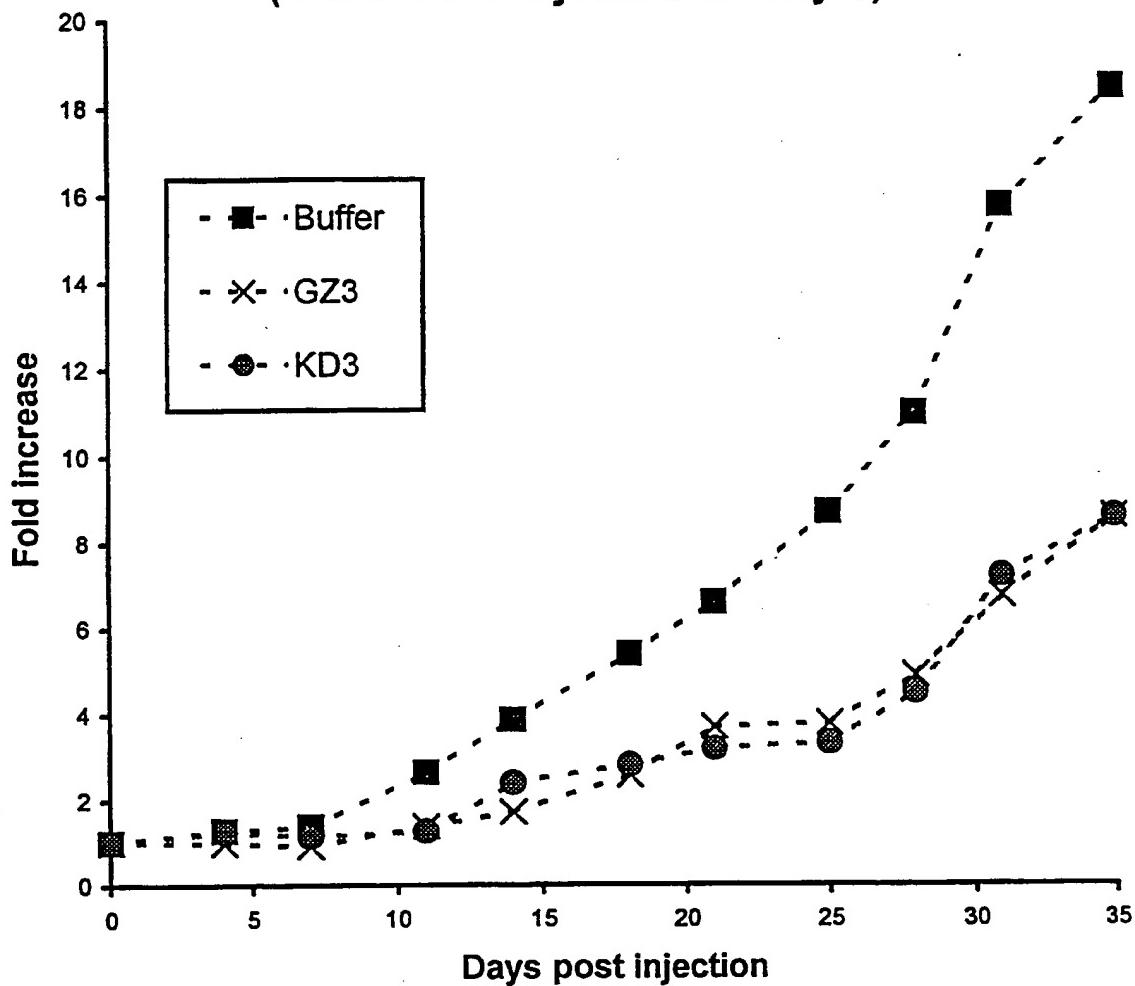


FIGURE 8B

9/16/00

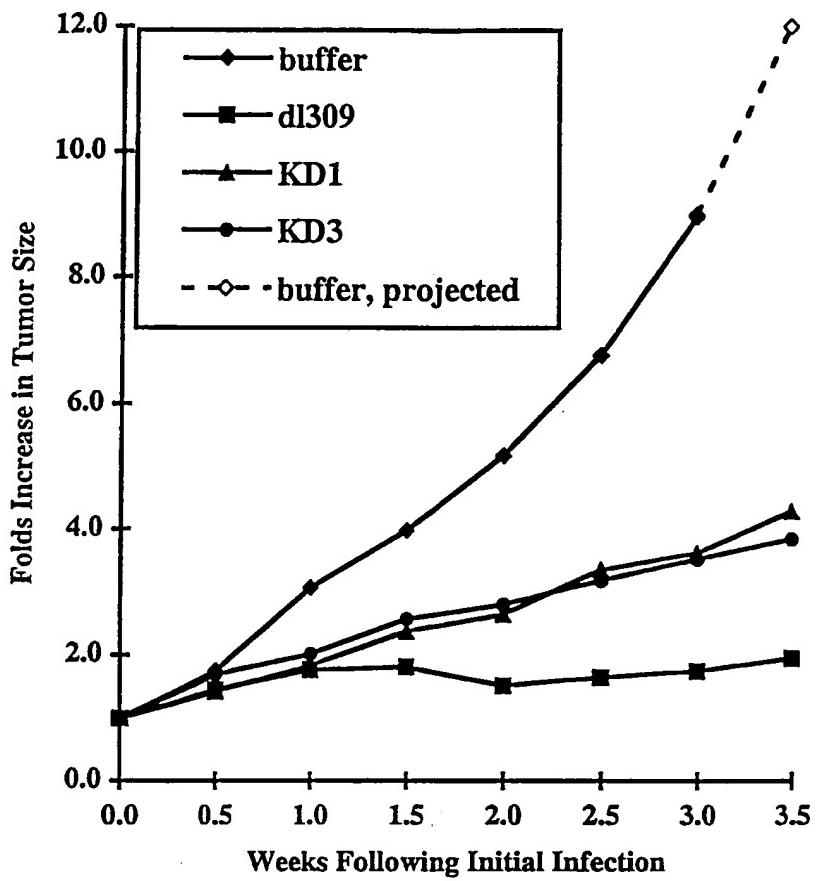


FIGURE 9

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Ad- β -gal Alone Ad- β -gal+*d*l01/07

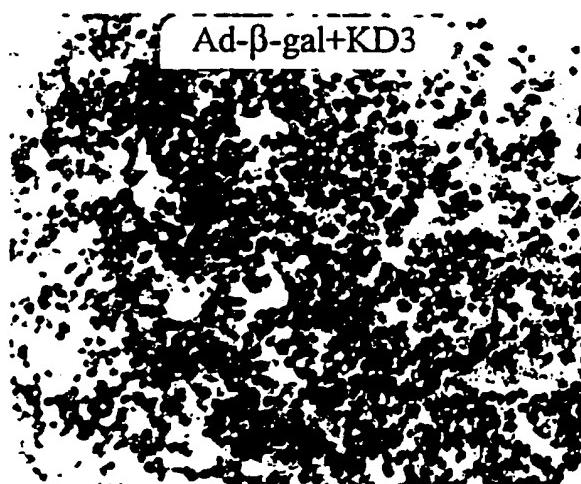
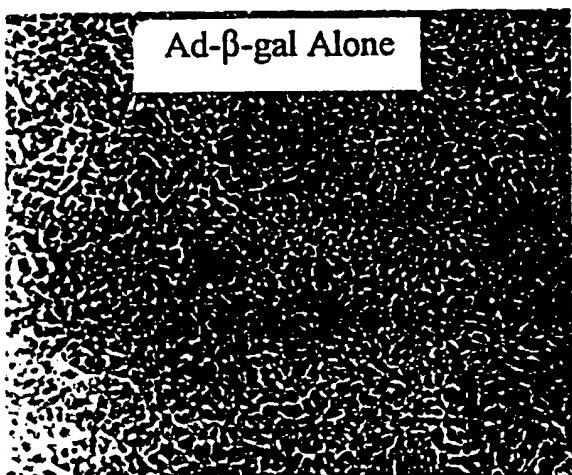
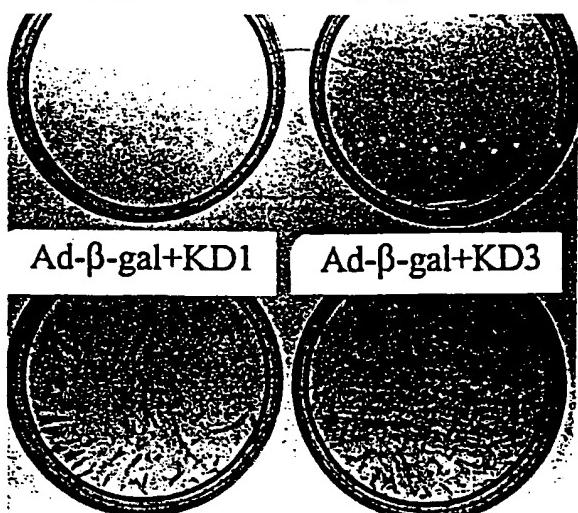


FIGURE 10

11/66

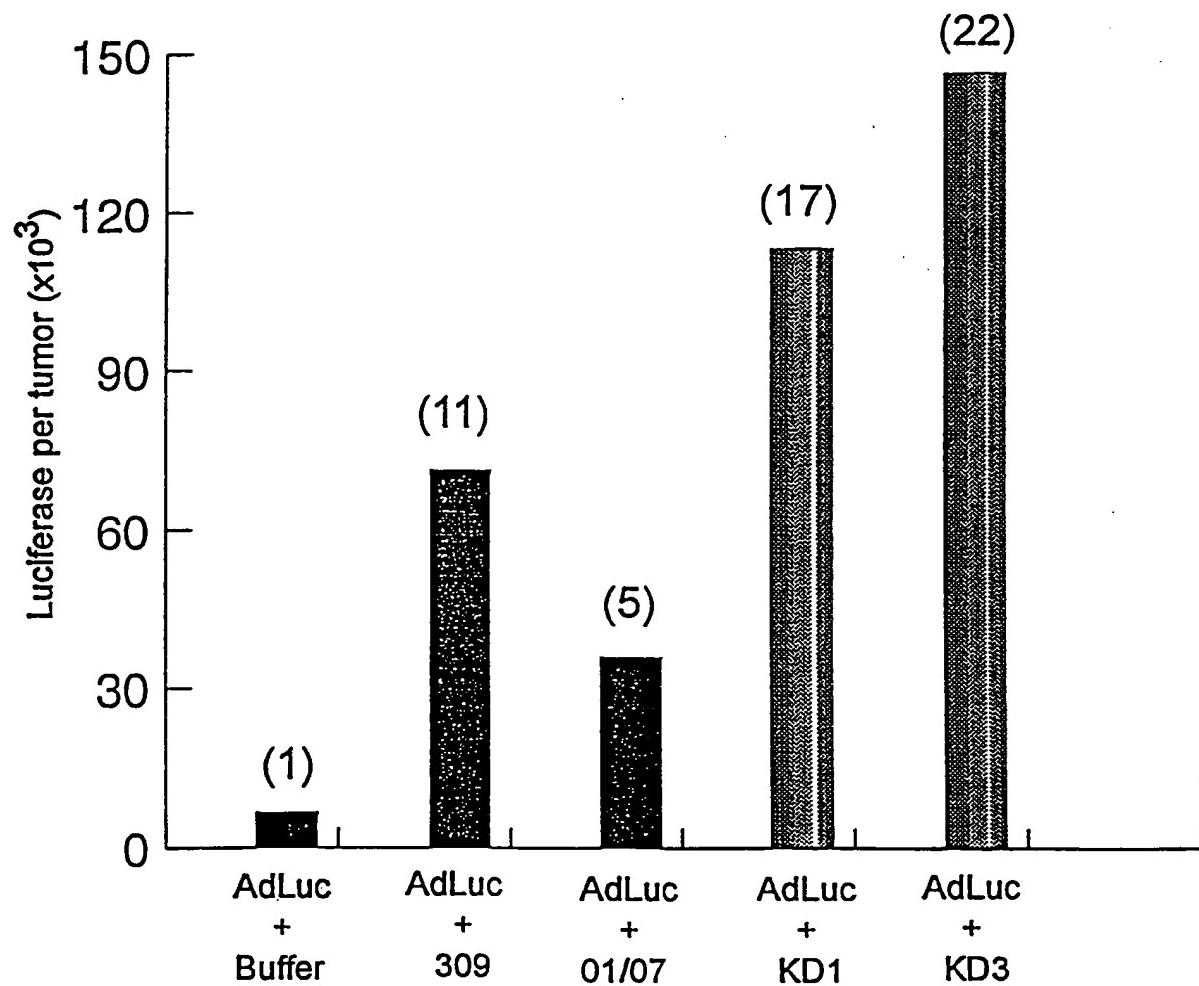


FIGURE 11

[Z]do

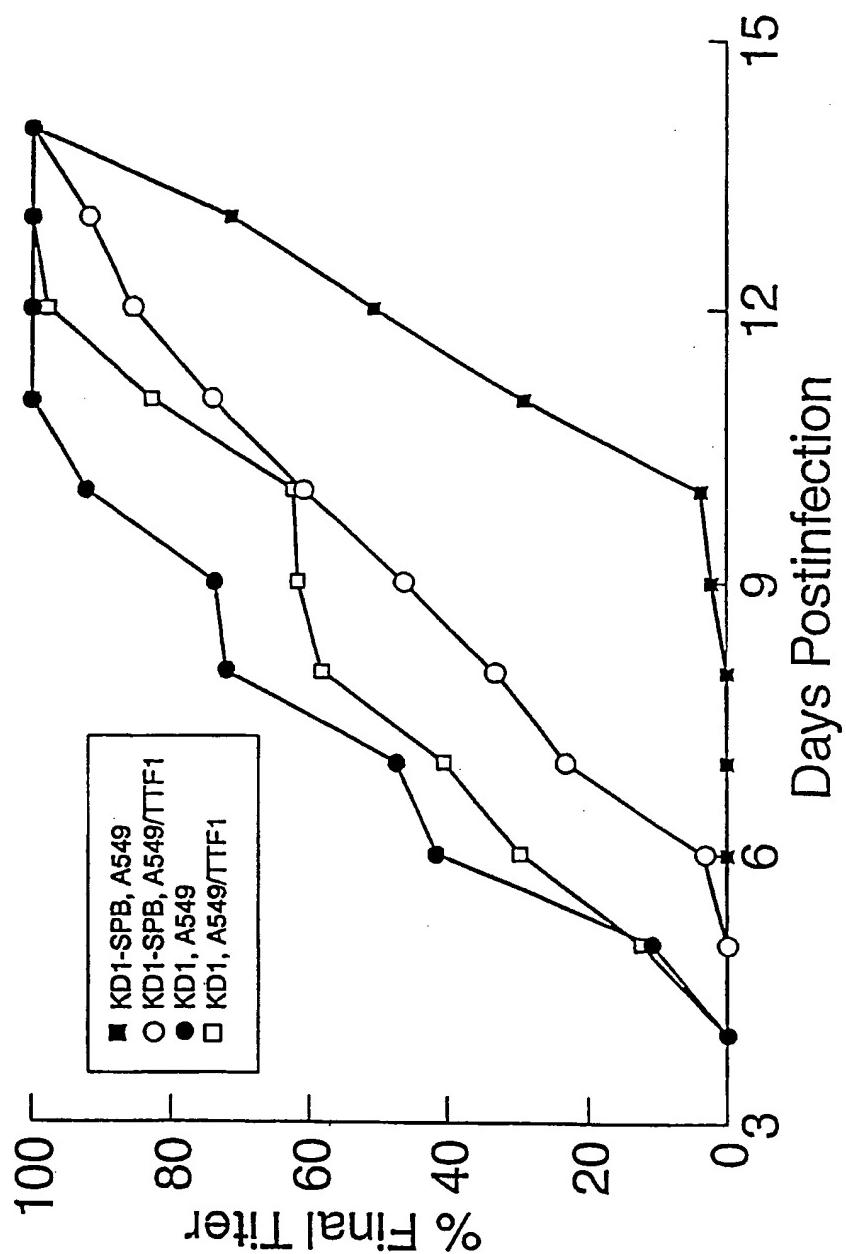


FIGURE 12

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KD1-SPB With the SPB Promoter in Place of the E4 Promoter Grows on H44a Lung Cancer Cells with the TTF1 Transcription Factor

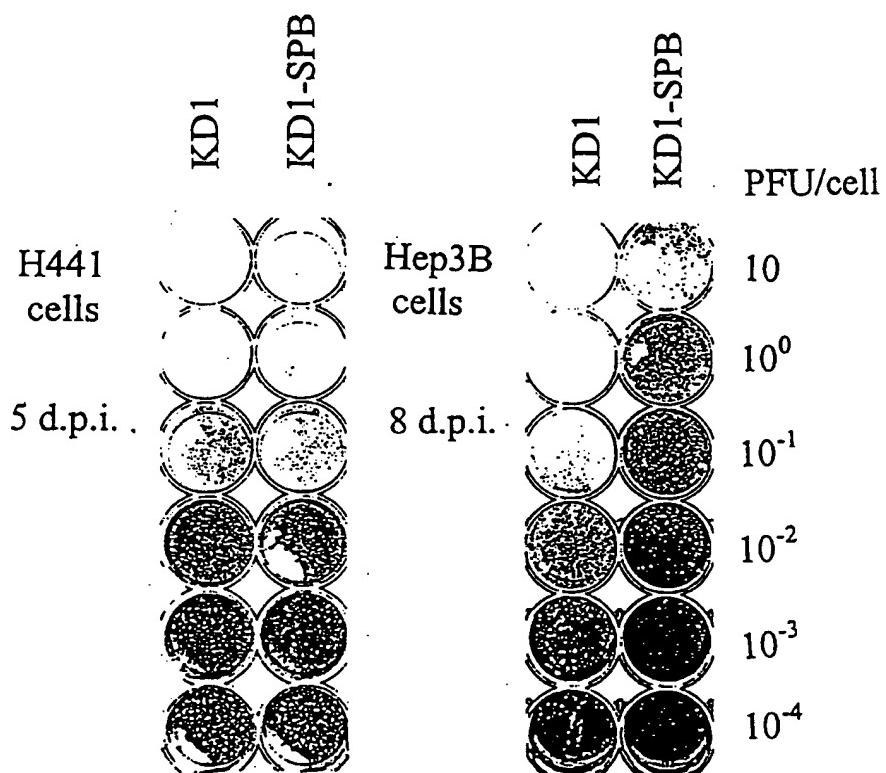


FIGURE 13

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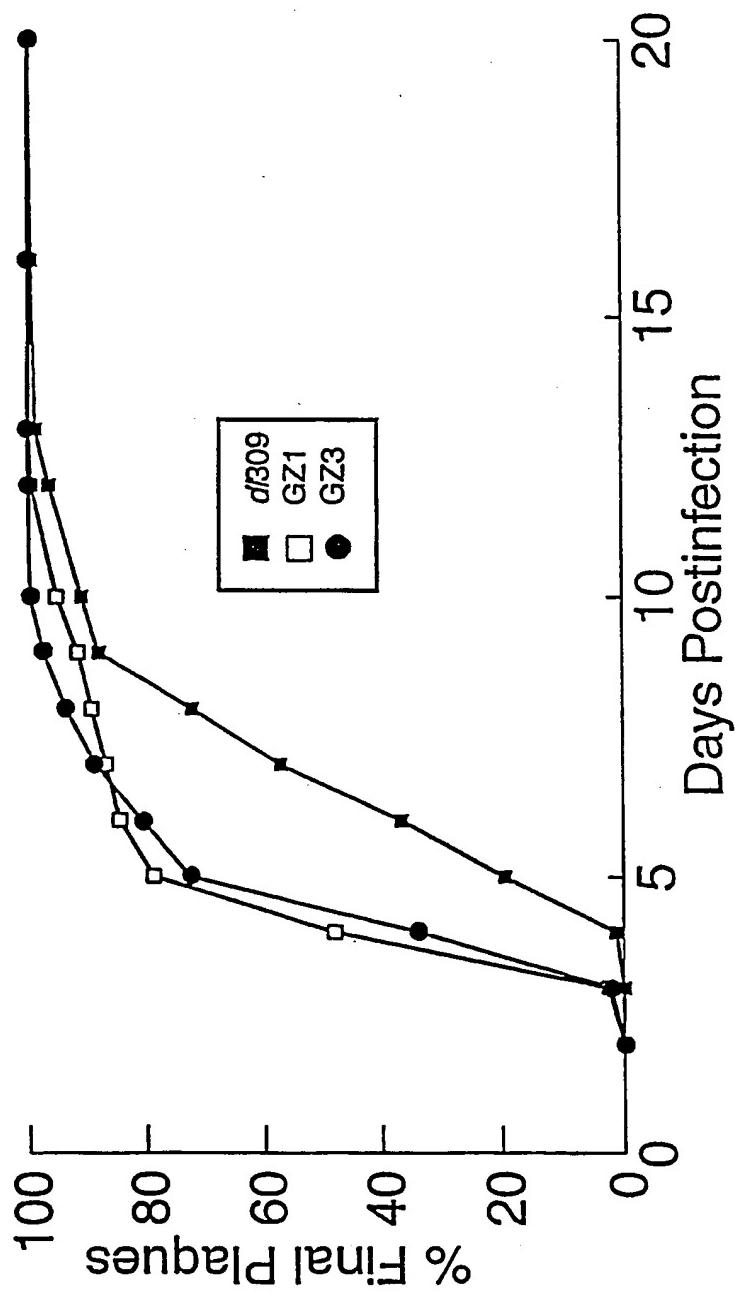


FIGURE 14

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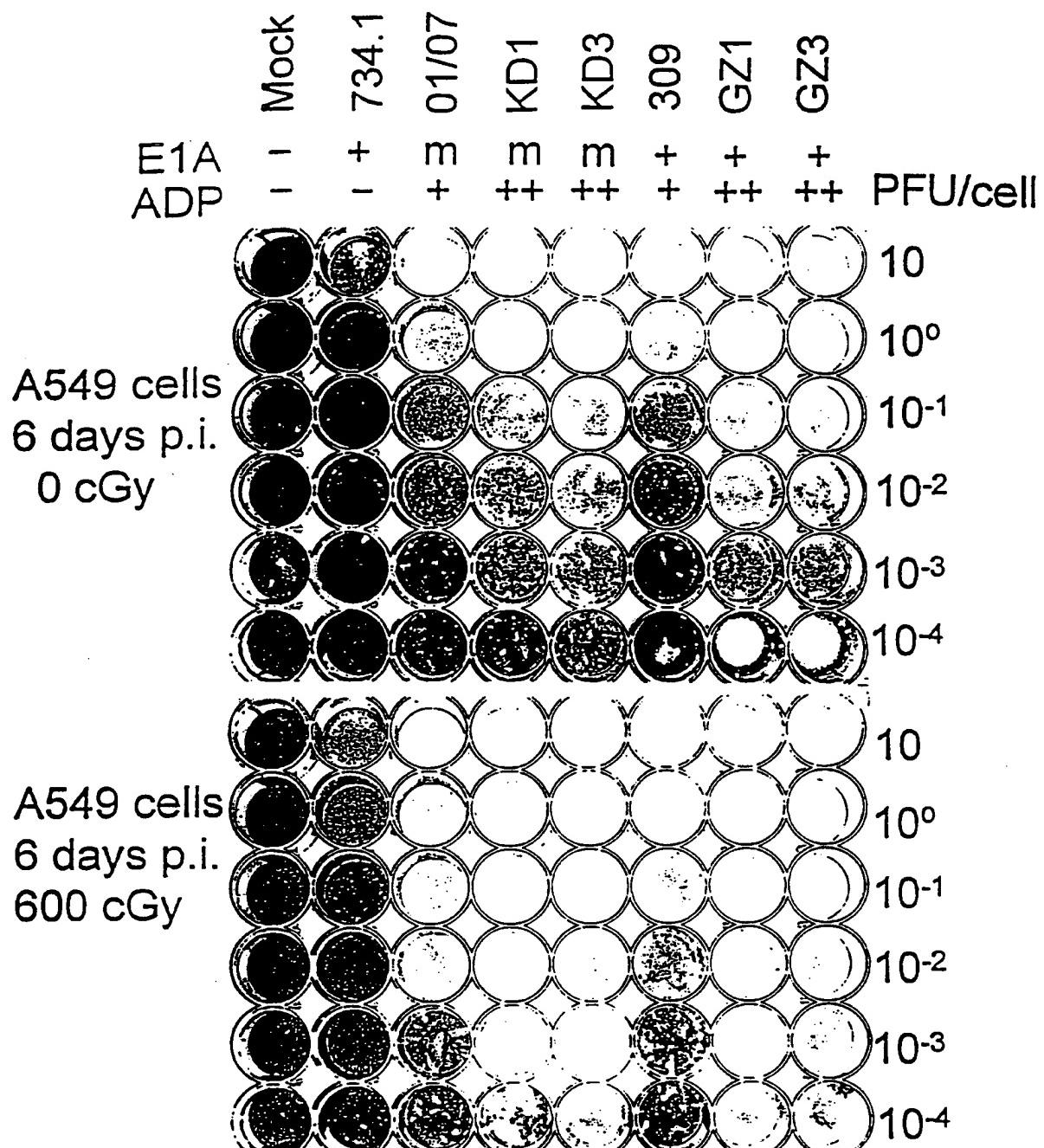


FIGURE 15

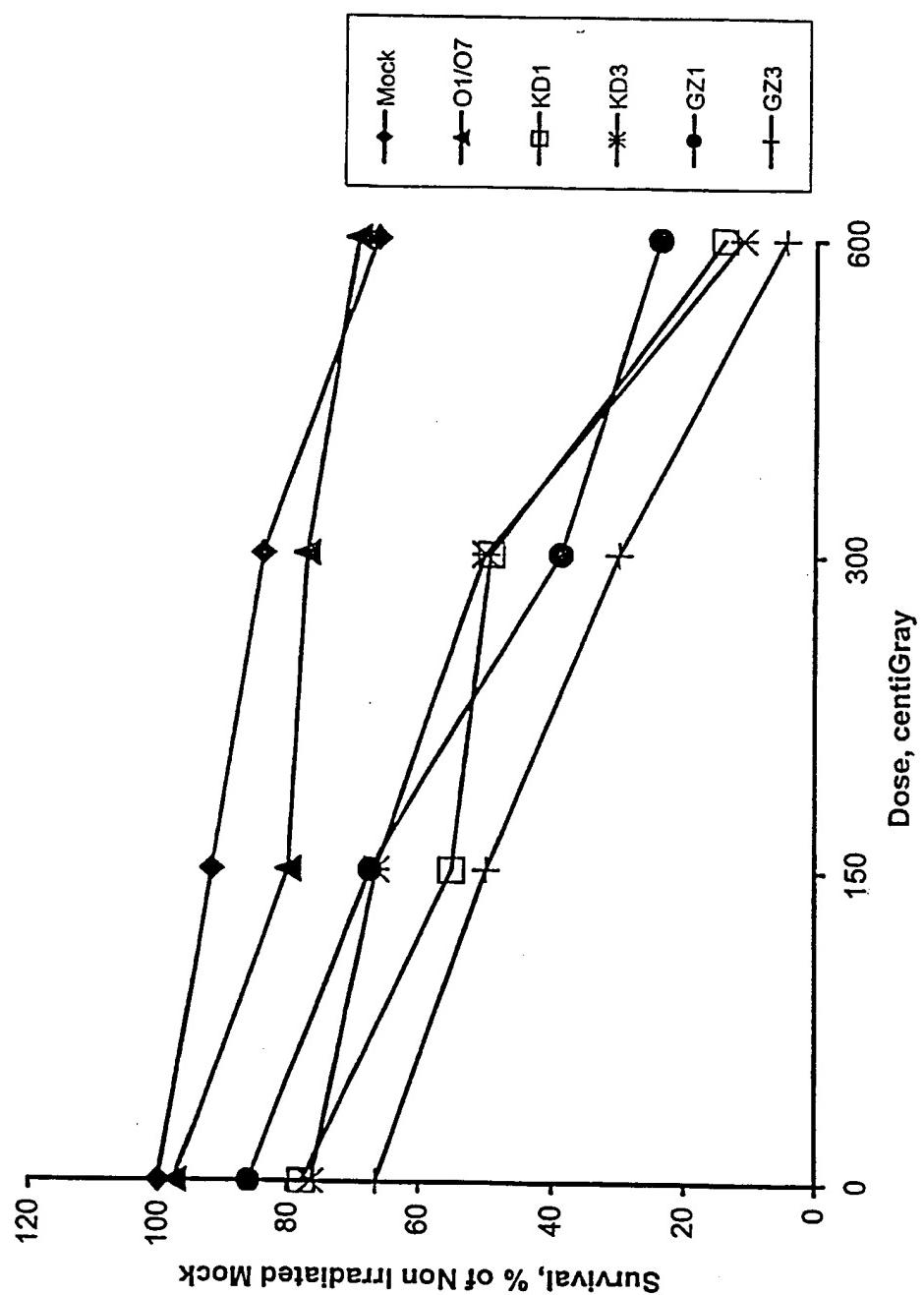


FIGURE 16

17166

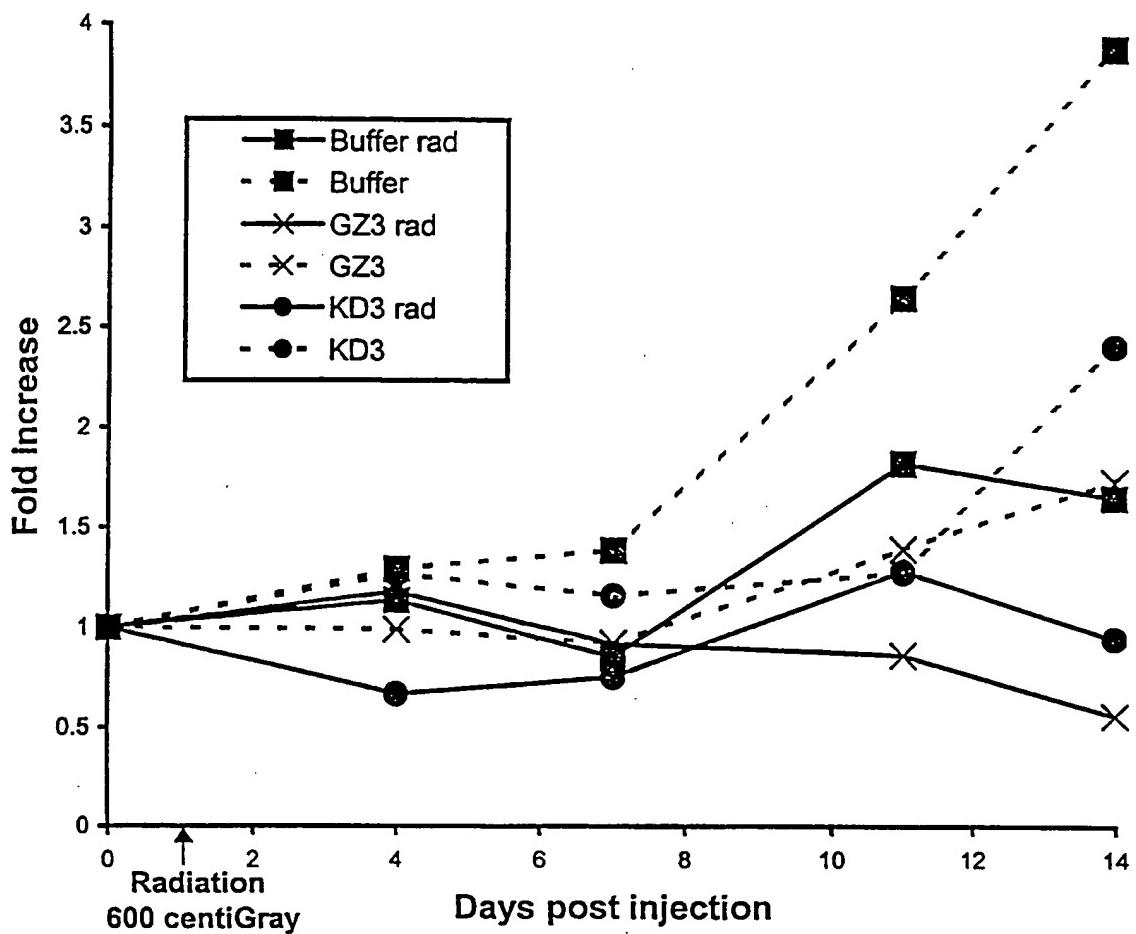


FIGURE 17

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Ad2 Adenovirus Death Protein

Luminal Domain

MIGSTIAPTTDYRNTTATGLTSANLPQVHAFVND 35

O - glycosylation N - glycosylation

WASLDMWWFSIALMFVCLIIMWLICCLKRRRARPP 70

|
*Transmembrane
(Signal - Anchor)*

|
Basic - Proline

IYRPIIVLNPHNEKIHRLDGLKPCSLLQYD 101

Cytoplasmic - Nucleoplasmic Domain

FIGURE 18A

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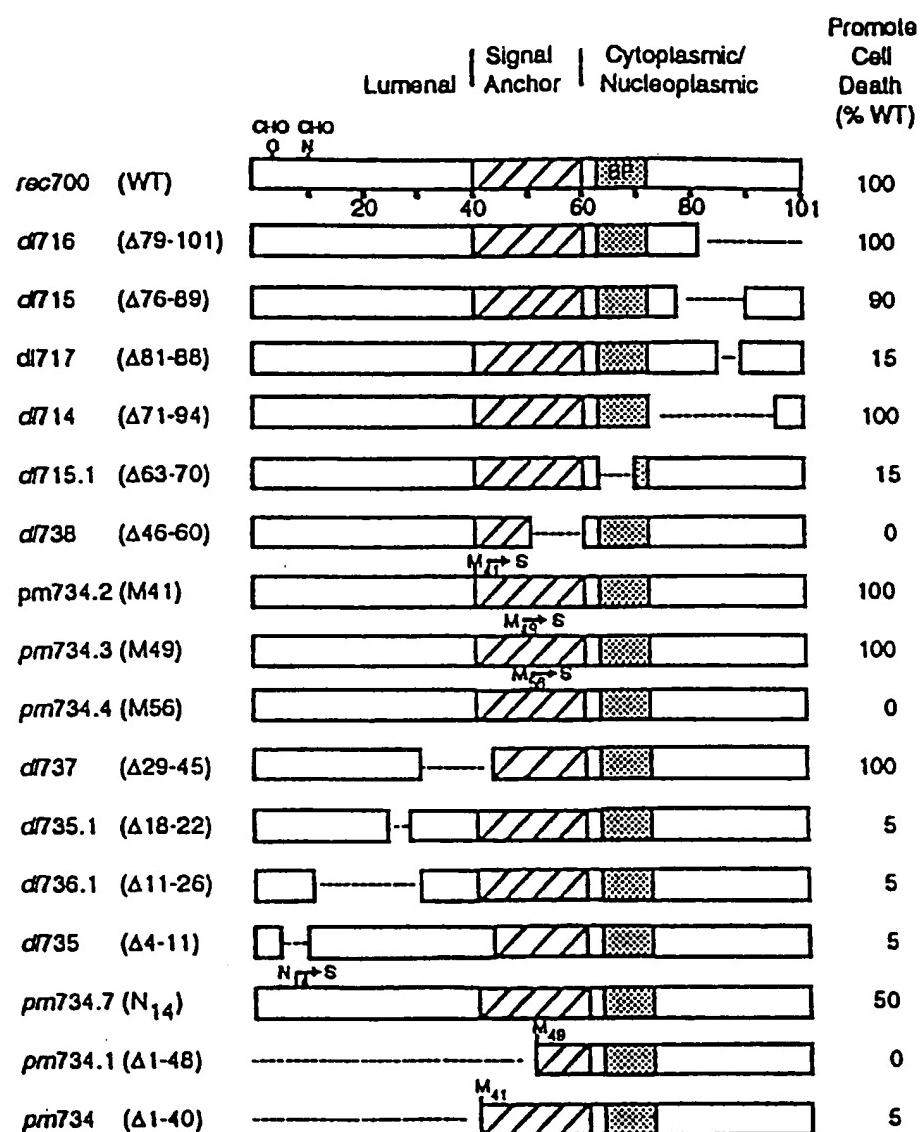


FIGURE 18B

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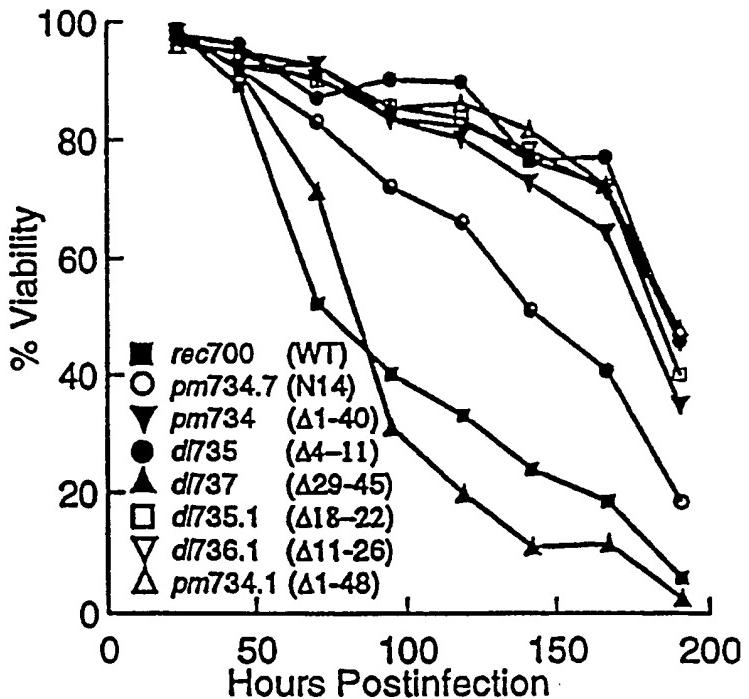


FIGURE 19A

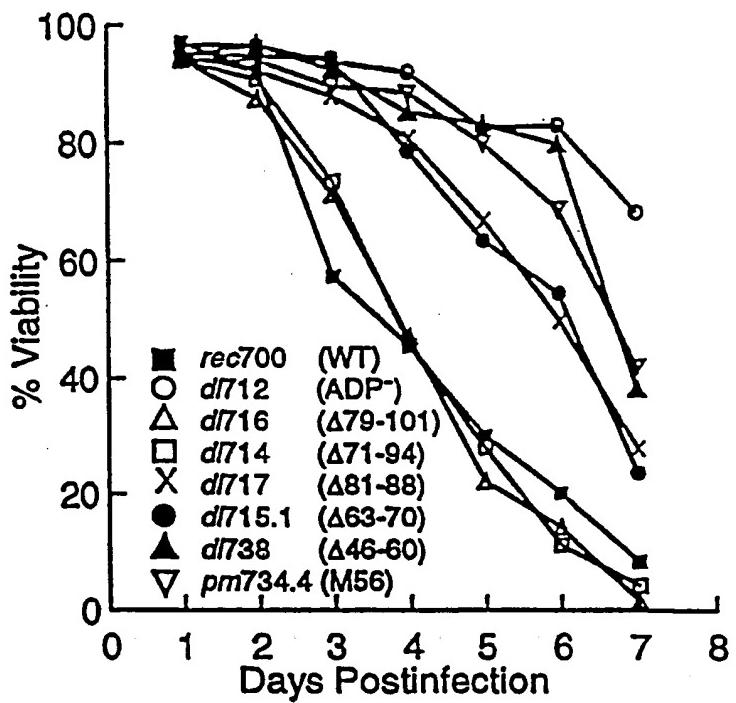


FIGURE 19B

Z1/66

Seq ID No.

	10	20	30	40	50
5 Ad1	-----MVDT VNSYNTATGL	TSALNLPQVS	TFVNNWANLG	MWWFSIALMF	
6 Ad2	MTGSTIAPTT DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF	
7 Ad5	-----MTN TTNAAAATGL	TSTTNTPQVS	AFVNNWDNLG	MWWFSIALMF	
8 Ad6	-----MVDT VNSYNTATGL	KSALNLPQVH	AFVNDWASLG	MWWFSIALMF	
9 dl716	MTGSTIAPTT DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF	
10 dl715	MTGSTIAPTT DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF	
11 dl714	MTGSTIAPTT DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	MWWFSIALMF	
12 dl737	MTGSTIAPTT DYRNTTATGL	TSALNLPQ--	-	-	IALMF
	60	70	80	90	100
5 Ad1	VCLIIIMWLSC	CLKRKRARPP	IYKPIIIVLNP	NNDGIHRLDG	LNTCSFSFAV -
6 Ad2	VCLIIIMWLIC	CLKRRRARPP	IYRPIIIVLNP	HNEKIHRLDG	LKPCSLLLQY D
7 Ad5	VCLIIIMWLIC	CLKRKRARPP	IYSPIIIVLHP	NNDGIHRLDG	LKHMFFSLTV -
8 Ad6	VCLIIIMWLIC	CLKRRRARPP	IYRPIIIVLNP	HNEKIHRLDG	LKPCSLLLQY D
9 dl716	VCLIIIMWLIC	CLKRRRARPP	IYRPIIIVL--	-	-
10 dl715	VCLIIIMWLIC	CLKRRRARPP	IYRPI-----	G	LKPCSLLLQY D
11 dl714	VCLIIIMWLIC	CLKRRRARPP	-----	SLLLQY	D
12 dl737	VCLIIIMWLIC	CLKRRRARPP	IYRPIIIVLNP	HNEKIHRLDG	LKPCSLLLQY D

Seq. ID No.

17 aa 1-40 of Ad2 ADP	MTGSTIAPTT DYRNTTATGL	TSALNLPQVH	AFVNDWASLD	
18 aa 41-59 of Ad2 ADP	MWWFSIALMF	VCLIIIMWLIC		
19 aa 63-70 of Ad2 ADP	KRRRARPP			
20 aa 60-101 of Ad2 ADP	C CLKRRRARPP	IYRPIIIVLNP	HNEKIHRLDG	LKPCSLLLQY D

FIGURE 20

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LOCUS ad5 comple 35935 bp DNA SYN 06-FEB-1999
 DEFINITION ad5 complete genome
 ACCESSION ad5 comple
 KEYWORDS .
 SOURCE Unknown.
 ORGANISM Unknown
 Unclassified.
 REFERENCE 1 (bases 1 to 35935)
 AUTHORS Self
 JOURNAL Unpublished.
 BASE COUNT 8367 a 10073 c 9761 g 7734 t
 ORIGIN

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 1 CATCATCAAT AATATAACCTT ATTTGGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
 61 TTGTGACGTG GCGCGGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
121 GATGTTGCAA GTGTGGCGGA ACACATGTA GCGACGGATG TGGCAAAAGT GACGTTTTG
181 GTGTGCGCCG GTGTACACAG GAAGTGACAA TTTTCGCGCG GTTTAGGCG GATGTTGTAG
241 TAAATTTGGG CGTAACCGAG TAAGATTGG CCATTTTCGC GGGAAAATG AATAAGAGGA
301 AGTGAAATCT GAATAATTTC GTGTTACTCA TAGCGCGTAA TATTTGCTA GGGCCGCGGG
361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTT CTCAAGGTGTT TTCCCGGTT
421 CGGGTCAAAG TTGGCGTTT ATTATTATAG TCAGCTGACG TGTAGTGTAT TTATACCCGG
481 TGAGTTCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCCGC
541 TCCGACACCG GGACTGAAAA TGAGACATAT TATCTGCCAC GGAGGTGTTA TTACCGAAGA
601 AATGGCCGCC AGTCTTTGG ACCAGCTGAT CGAAGAGGTA CTGGCTGATA ATCTTCCACC
661 TCCTAGCCAT TTGAAACCAC CTACCCCTCA CGAACTGTAT GATTTAGACG TGACGGCCCC
721 CGAAGATCCC AACGAGGAGG CGGTTTCGCA GATTTTTCCC GACTCTGTA TGTTGGCGGT
781 GCAGGAAGGG ATTGACTTAC TCACTTTTC GCGGGCGCCC GTTCTCCCG AGCCGCCTCA
841 CCTTTCCCGG CAGCCCGAGC AGCCGGAGCA GAGAGCCTTG GTTCCGGTTT CTATGCCAAA
901 CCTTGTACCG GAGGTGATCG ATCTTACCTG CCACGAGGCT GGCTTTCCAC CCAGTGACGA
961 CGAGGATGAA GAGGGTGAGG AGTTTGTT AGATTATGTG GAGCACCCCC GGCACGGTTG
1021 CAGGTCTTGT CATTATCACC GGAGGAATAC GGGGGACCCA GATATTATGT GTTCCGTTTG
1081 CTATATGAGG ACCTGTGGCA TGTGTTGTCTA CAGTAAGTGA AAATTATGGG CAGTGGGTGA
1141 TAGAGTGGTG GTTTGGGTGT GGTAATTTTT TTITTAATT TTACAGTTT GTGGTTAAA
1201 GAATTTTGTA TTGTGATTTT TTAAAAGGT CCTGTGTCG AACCTGAGCC TGAGCCCGAG
1261 CCAGAACCGG AGCCTGCAAG ACCTACCCCG CGTCTCTAAA TGGCGCCTGC TATCTGAGA
1321 CGCCCGACAT CACCTGTGTC TAGAGAATGC AATAGTAGA CGGATAGCTG TGACTCCGGT
1381 CCTCTTAACA CACCTCCCTGA GATACACCCG GTGGTCCCGC TGTGCCCCAT TAAACCAGTT
1441 GCCGTGAGAG TTGGTGGCGC TCGCAGGCT GTGAAATGTA TCGAGGACTT GCTTAACGAG
1501 CCTGGCAAC CTTGGACTT GAGCTGAAA CGCCCCAGGC CATAAGGTGT AAACCTGTGA
1561 TTGCGTGTGT GTTAAACGCC TTTGTTGCT GAATGAGTTG ATGTAAGTTT AATAAAGGGT
1621 GAGATAATGT TAACTGCA TGGCGTGTAA AATGGGGCGG GGCTTAAAGG GTATATAATG
1681 CGCCGTGGGC TAATCTGGT TACATCTGAC CTCATGGAGG CTTGGGAGTG TTTGGAAGAT
1741 TTTCTGCTG TCGTAACCTT GCTGGAACAG AGCTCTAACAA GTACCTCTTG GTTTGGAGG
1801 TTTCTGTGGG GCTCATCCCA GGCAGGTTA GTCTGCAGAA TTAAGGAGGA TTACAAGTGG
1861 GAATTGAAAG AGCTTTGAA ATCCTGTGGT GAGCTGTTG ATTCTTTGAA TCTGGGTAC
1921 CAGGCCTTT TCCAAGAGAA GGTCAATCAAG ACTTTGGATT TTTCCACACC GGGGCGCGCT
1981 GCGGCTGCTG TTGCTTTTTT GAGTTTATA AAGGATAAAAT GGAGCGAAGA AACCCATCTG
2041 AGCGGGGGGT ACCTGCTGGA TTTCTGGCC ATGCATCTGT GGAGAGCGGT TGTGAGACAC
2101 AAGAATCGCC TGCTACTGTT GTCTTCCGTC CGCCCGGCCA TAATACCGAC GGAGGAGCAG
2161 CAGCAGCAGC AGGAGGAAGC CAGGCGCGG CGGCAGGAGC AGAGCCCATG GAACCCGAGA
2221 GCGGGCCTGG ACCCTCGGGA ATGAATGTTG TACAGGTGGC TGAACGTAT CCAGAACTGA
2281 GACGCATTTT GACAATTACA GAGGATGGC AGGGGCTAAA GGGGGTAAAG AGGGAGCGGG
2341 GGGCTTGTGA GGCTACAGAG GAGGCTAGGA ATCTAGCTTT TAGCTTAATG ACCAGACACC
2401 GTCTGAGTG TTTACTTTT CAACAGATCA AGGATAATTG CGCTAATGAG CTTGATCTGC
2461 TGGCGAGAA GTATTCCATA GAGCAGTGA CCACCTACTG GCTGCAGCCA GGGGATGATT
2521 TTGAGGAGGC TTTAGGGTA TATGCAAAGG TGGCACTTAG GCCAGATTGC AAGTACAAGA
2581 TCAGCAAACG TGAAATATC AGGAATTGTT GCTACATTTC TGGGAACGGG GCCGAGGTGG
2641 AGATAGATAC GGAGGATAGG GTGGCCTTTA GATGTAGCAT GATAAAATATG TGGCCGGGGG
  
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ad5

FIGURE 21
(SHEET 1)

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2701 TGCTTGGCAT GGACGGGGTG GTTATTATGA ATGTAAGGTT TACTGGCCCC AATTTAGCG
 2761 GTACGGTTTT CCTGGCCAAT ACCAACCTTA TCCTACACGG TGTAAGCTTC TATGGGTTTA
 2821 ACAATACTG TGTGGAAGCC TGGACCGATG TAAGGGTCG GGGCTGTGCC TTTTACTGCT
 2881 GCTGGAAGGG GGTGGTGTGT CGCCCCAAA GCAGGGCTTC ATTAAAGAAA TGCCTCTTIG
 2941 AAAGGTGTAC CTTGGGTATC CTGTCTGAGG GTAACCTCAG GGTGCGCCAC AATGTGGCCT
 3001 CCGACTGTGG TTGCTTCATG CTAGTGAAAA CGCTGGCTGT GATTAAGCAT AACATGGTAT
 3061 GTGGCAACTG CGAGGACAGG GCCTCTCAGA TGCTGACCTG CTCGGACGGC AACTGTCACC
 3121 TGCTGAAGAC CATTCACTGTA GCGACCACT CTCGCAAGGG CTGGCCAGTG TTTGAGCATA
 3181 ACATACTGAC CCGCTGTTCC TTGCAATTGG GTAAACAGGAG GGGGGTGTTC CTACCTTACC
 3241 AATGCAATTG GAGTCACACT AGATATTGTC TTGAGCCCAGA GAGCATGTTCC AAGGTGAACC
 3301 TGAACGGGGT GTTGTGACATG ACCATGAAAGA TCTGGAAGGT GCTGAGGTAC GATGAGACCC
 3361 GCACCAAGGT CAGACCTGCG GAGTGTGGCG GTAAACATAT TAGGAACCAAG CCTGTGATGC
 3421 TGGATGTGAC CGAGGAGCTG AGGCCCCGATC ACTTGGTGCT GGCCTGCAAC CGCGCTGAGT
 3481 TTGGCTCTAG CGATGAAGAT ACAGATTGAG GTACTGAAAT GTGTTGGCGT GGCTTAAGGG
 3541 TGGGAAAGAA TATATAAGGT GGGGGTCTTA TGTAGTTTG TATCTGTTT GCAGCAGCCG
 3601 CCGCCGCCAT GAGCACCAAC TCGTTTGATG GAAGCATTGT GAGCTCATAT TTGACAACGC
 3661 GCATGCCCTT ATGGGCCGGG GTGCGTCAGA ATGTAATGGG CTCCAGCATT GATGGTCGCC
 3721 CCGTCCTGCC CGCAAACCTCT ACTACCTTGA CCTACGAGAC CGTGTCTGGA ACGCCGTTGG
 3781 AGACTGAGC CTCCGCCGCC GCTTCAGCCG CTGCAAGCCAC CGCCCGCGGG ATTGTGACTG
 3841 ACTTTGCTT CCTGAGCCCC GCACAATTGG ATTCTTGTAC CCGGGAACTT AATGTCGTTT
 3901 ACAAGTTGAC GGCTCTTTG CGCCAGCAGG TTTCTGCCCT GAAGGCTTCC TCCCCCTCCA
 3961 CTCAGCAGCT GTTGGATCTG AAAAAACCAAG ACTCTGTTTG GATTTGGATC AAGCAAGTGT
 4021 ATGCGGTTTA AAACATAAT AAAAAACCAAG ACTCTGTTTG GATTTGGATC AAGCAAGTGT
 4081 CTTGCTGTCT TTATTTAGGG GTTTTGGCG CGCGGTAGGC CCGGGACCAG CGGTCTCGGT
 4141 CGTTGAGGGT CCTGTGTATT TTTTCCAGGA CGTGGTAAAG GTGACTCTGG ATGTTAGAT
 4201 ACATGGGCAT AAGCCCGTCT CTGGGGTGGG GGTAGCACCA CTGCAGAGCT TCATGCTGCG
 4261 GGGTGGTGT GTAGATGATC CAGTCGTAGC AGGAGCGCTG GGCGTGGTGC CTAAAAATGT
 4321 CTTTCAGTAG CAAGCTGATT GCCAGGGCA GGGCCTTGGT GTAAGTGTAAACAAAGCGGT
 4381 TAAGCTGGGA TGGGTGCATA CGTGGGGATA TGAGATGCAT CTTGGACTGT ATTTTTAGGT
 4441 TGGCTATGTT CCCAGCCATA TCCCTCCGGG GATTCACTGTT GTGAGAACCC ACCAGCACAG
 4501 TGTATCCGGT GCACCTGGGA AATTTCATGTT GTAGCTTAAAGA AGGAAATGCG TGGAAAGAACT
 4561 TGGAGACGCC CTTGTCACCT CCAAGATTT CCATGCATTG GTCCATAATG ATGCCATATGG
 4621 GCCCACGGGC GGCGGCCCTGG GCGAAGATAT TTCTGGGATC ACTAACGTCA TAGTTGTGTT
 4681 CCAGGATGAG ATCGTCATAG GCCATTGTTA CAAAGCGCGG GCGGAGGGTG CCAGACTGCG
 4741 GTATAATGGT TCCATCCGGC CCAGGGCGT AGTACCCCTC ACAGATTGCA ATTTCACCG
 4801 CTTTGAGTTC AGATGGGGGG ATCATGTCATA CCTGCGGGGC GATGAAGAAA ACGGTTTCCG
 4861 GGGTAGGGGA GATCAGCTGG GAAGAAAGCA GTTCTGGAG CAGCTGCGAC TTACCGCAGC
 4921 CGGTGGGCC GAAATCACA CCTATTACCG GGTGCAACTG GTAGTTAAGA GAGCTGCAGC
 4981 TGCCGTATC CCTGAGCAGG GGGGCCACTT CGTTAACGCAT GTCCCTGACT CGCATGTTT
 5041 CCCTGACCAA ATCCGCCAGA AGGCCTCGC CGCCCGAGCGA TAGCAGTTCT TGCAAGGAAG
 5101 CAAAGTTTT CAACGGTTTG AGACCGTCGG CGTGTAGGCAT GCTTTGAGC GTTTGACCAA
 5161 GCAGTCCAG GCGGTCCCAC AGCTCGGTCA CCTGCTCTAC GGCATCTCGA TCCAGCATAT
 5221 CTCCTCGTTT CGCGGGTTGG GGCGGCTTC GCTGTACGGC AGTAGTCCGGT GTCAGCTGAC
 5281 ACGGGCCAGG GTCATGTCTT TCCACGGCG CAGGGTCTC GTCAGCTGAG TCTGGTCAC
 5341 GGTGAAGGGG TCGCTCCGG GCTGCGCGCT GGCCAGGGTG CGCTTGAAGGC TGGTCTGCT
 5401 GGTGCTGAAG CGCTGCCGGT CTTCGCCCTG CGCGTCCGGC AGGTAGCATT TGACCATGGT
 5461 GTCATAGTCC AGCCCCCTCCG CGCGTGGCC CTTGGCGCGC AGCTTGCCT TGGAGGAGGC
 5521 GCGCAGCAGAG GGGCAGTGCAGA GACTTTTGAG GCGTAGAGC TTGGGCGGA GAAATACCGA
 5581 TTCCGGGGAG TAGGCATCCG CGCCAGGGC CCGCGAGACG GTCTCGCATT CCACGAGCCA
 5641 GGTGAGCTCT GGGCGTTCGG GGTCAAAAC CAGGTTTCCC CCATGTTT TGATGCGTTT
 5701 CTTACCTCTG GTTCCATGA GCGGGTGTCC ACGCTCGGTG ACGAAAAGGC TGTCCGTGTC
 5761 CCCGTATACA GACTTGAGAG GCCTGTCCTC GAGCGGTGTT CGCGGTCTC CCTCGTATAG
 5821 AAACTCGGAC CACTCTGAGA CAAAGGCTCG CGTCCAGGCC AGCACGAAGG AGGCTAAGTG
 5881 GGAGGGGGTAG CGGTGTTGT CCAGTGGGG GTCACTCGC TCCAGGGTGT GAAGACACAT
 5941 GTGCCCTCT TCGGCATCAA GGAAGGTGAT TGGTTGTAG GTGTTAGGCC CGTGACCGGG
 6001 TGTTCTGAA GGGGGCTAT AAAAGGGGGT GGGGGCGCGT TCGTCCTCAC TCTCTCCCGC
 6061 ATCGCTGTCT GCGAGGGCCA GCTGTTGGGG TGAGTACTCC CTCTGAAAAG CGGGCATGAC

6121 TTCTGCGCTA AGATTGTCAG TTTCCAAAAA CGAGGGAGGAT TTGATAATTCA CCTGGCCCCGC
 6181 GGTGATGCCCT TTGAGGGTGG CCGCATCCAT CTGGTCAGAA AAGACAATCT TTTTGTGTC
 6241 AAGCTTGGTG GCAAACGACC CGTAGAGGGC GTTGGACAGC AACTTGGCGA TGGAGCGCAG
 6301 GGTTTGGTTT TTGTCGCGAT CGGCGCGCTC CTTGGCCGCG ATGTTTAGCT GCACGTATTC
 6361 GCGCGCAACG CACCGCCATT CGGGAAAGAC GGTGGTGCAC TCGTCGGCA CCAGGTGCAC
 6421 GCGCCAACCG CGGTTGTGCA GGGTGACAAG GTCAACGCTG GTGGCTACCT CTCCGCGTAG
 6481 GCGCTCGTTG GTCCAGCAGA GGCGGCCGCG CTTGCGCGAG CAGAATGGCG TAGGGGGTC
 6541 TAGCTCGCTC TCGTCCGGGG GGTCTGCGTC CACGGTAAAG ACCCCGGGCA GCAGGCGCGC
 6601 GTCGAAGTAG TCTATCTTGC ATCCTGCAA GTCTAGCGCC TGCTGCCATG CGCGGGCGGC
 6661 AAGCGCGCGC TCGTATGGGT TGAGTGGGG ACCCCATGGC ATGGGGTGGG TGAGCGCGGA
 6721 GCGTACATG CGCCTAACATGT CGTAAACGTA GAGGGCTCT CTGAGTATTC CAAGATATGT
 6781 AGGGTAGCAT CTTCACCGC GGATGCTGGC GCGCACGTA TCGTATAGTT CGTGCAGGG
 6841 AGCGAGGAGG TCGGGACCGA GTTGTACG GGCGGGCTGC TCTGCTCGA AGACTATCTG
 6901 CCTGAAGATG GCATGTGAGT TGGATGATAT GTTGGACGC TGGAAAGACGT TGAAGCTGGC
 6961 GTCTGTGAGA CCTACCGCGT CACGCACGAA GGAGGCGTAG GAGTCGCGCA GCTTGTGAC
 7021 CAGCTCGCGC GTGACCTGCA CGTCTAGGGC GCAGTAGTCC AGGGTTTCT TGATGATGTC
 7081 ATACTTATCC TGTCCCTTTT TTTTCCACAG CTGCGGTG AGGACAAAAGT CTTGGCGTC
 7141 TTTCCAGTAC TCTTGGATCG GAAACCGTC GGCCTCCGAA CGGTAAGAGC CTAGCATGTA
 7201 GAACTGGTTG ACGGCCTGGT AGGCGCAGCA TCCCCTTTCT ACGGGTAGCG CGTATGCCG
 7261 CGCGGCCCTTC CGGAGCGAGG TGTGGGTGAG CGCAAAGGTG TCCCTGACCA TGACTTTGAG
 7321 GTACTGGTAT TTGAAGTCAG TGTCTCGCA TCCGCCCTGC TCCCAGAGCA AAAAGTCCGT
 7381 GCGCTTTTTG GAACGCGGAT TTGGCAGGGC GAAGGTGACA TGTTGAAGA GTATCTTCC
 7441 CGCGCGAGGC ATAAGTTGCG GTGTGATGCG GAAGGGTCCC GGCACCTCGG AACGGTTGTT
 7501 AATTACCTGG GCGCGAGCA CGATCTCGTCA AAAGCGGTG ATGTTGTGGC CCACAATGTA
 7561 AAGTTCCAAG AAGCGGGGGA TGCCCTTGT GGAAAGGCAAT TTTTTAAGTT CCTCGTAGGT
 7621 GAGCTCTTCA GGGGAGCTGA GCGCGTGC TGAAAGGGC CAGTCTGCAA GATGAGGTT
 7681 GGAAGCGACG AATGAGCTCC ACAGGTGACG GGCCATTAGC ATTTCGAGGT GGTGCGAAA
 7741 GGTCTAACAC TGCGACCTA TGCCATTGTT TTCTGGGTG ATGAGTAGA AGGTAAGCGG
 7801 GTCTTGTTC CAGGGCTCCC ATCCAAGGTT CGCGCTAGG TCTCGCGCG CAGTCACTAG
 7861 AGGCTCATCT CGCGCGAACT TCATGACAG CATGAAGGGC ACAGAGCTGCT TCCCAAAGGC
 7921 CCCATCCAA GTATAGGTCT CTACATCGTA GGTGACAAAG AGACGCTCGG TGCGAGGATG
 7981 CGAGCCGATC GGGAAAGAACT GGATCTCCCG CCACCAATTG GAGGAGTGGC TATTGATGTC
 8041 GTGAAAGTAG AAGTCCCTGC GACGGGGCGA ACACCTGTC TGGCTTTGT AAAAACGTGC
 8101 GCAGTACTGG CAGCGGTGCA CGGGCTGTAC ATCTGCAAG AGGTTGACCT GACGACCGCG
 8161 CACAAGGAAG CAGAGTGGGA ATTTGAGCCC CTGGCCTGGC GGGTTTGGCT GGTGGCTTC
 8221 TACTTCGGCT GCTTGTCTT GACCGTCTGG CTGCTCGAGG GGAGTTACGG TGGATCGGAC
 8281 CACCAACGCC CGCGAGCCCA AAGTCCAGAT GTCCCGCGC GCGGGTCCGA GCTTGATGAC
 8341 AACATCGCGC AGATGGGAGC TGTCCATGGT CTGGAGCTCC CGGGCGTCA GGTCAAGGCGG
 8401 GAGCTCTGCA AGGTTTACCT CGCATAGACG GGTCAAGGGCG CGGGCTAGAT CCAGGTGATA
 8461 CCTAATTTC CAGGGCTGGT TGGTGGCGC GTGATGGCT TGCAAGAGGC CGCATCCCCG
 8521 CGGCGCGACT ACGGTACCGC GCGCGGGCG GTGGGCCGCG GGGGTGTCCT TGGATGATGC
 8581 ATCTAAAAGC GGTGACGCGG GCGAGCCCCC GGAGGTAGGG GGGGCTCCGG ACCCGCCGGG
 8641 AGAGGGGGCA GGGGCACGTC GCGCGCGC GCGGGCAGGA GCTGGTGTG CGCGCGTAGG
 8701 TTGCTGGCGA ACAGCGACGAC GCGCGGTG ATCTCCTGAA TCTGGCGCT CTGCGTGAAG
 8761 ACGACGGGCC CGGTGAGCTT GAGCCTGAAA GAGAGTTCGA CAGAATCAAT TTCGGTGTG
 8821 TTGACGGCGG CCTGGCGAA AATCTCTGC ACGTCCTG AGTTGTCTTG ATAGGCGATC
 8881 TCGGCCATGA ACTGCTCGAT CTCTTCTCTC TGGAGATCTC CGCGTCCCGC TCGCTCCACG
 8941 GTGGCGCGA GGTGTTGGA AATGCGGGCC ATGAGCTGCG AGAAGGCGTT GAGGCGCTCCC
 9001 TCGTCCAGA CGCGCTGTA GACCACGCC CCTCGGGCAT CGCGGGCGC CATGACCAACC
 9061 TGCGCGAGAT TGAGCTCCAC GTGCCGGCG AAGACGGCGT AGTTTCGAG GCGCTGAAAG
 9121 AGGTAGTTGA GGGTGGTGGC GGTGTGTTCT GCCACGAAGA AGTACATAAC CCAGCGTCGC
 9181 AACGTGGATT CGTTGATATC CCCCCAAGGCC TCAAGGCGCT CCATGGCCTC GTAGAAGTCC
 9241 ACGGCGAAGT TGAAAAAACTG GGAGTGTGCG GCCGACACGG TTAACTCCTC CTCCAGAAGA
 9301 CGGATGAGCT CGCGACAGT GTCGCGACCC TCGCGCTCAA AGGCTACAGG GGCCCTTCT
 9361 TCTTCTTCA TCTCCTCTC CATAAGGGCC TCCCTTCTT CTTCCTCTGG CGGGCGTGGG
 9421 GGAGGGGGGA CACGGCGGGC ACGACGGCG ACCGGGAGGC GTGACGGCGC GGCGTCTC
 9481 ATCTCCCCGC GCGACGGCG CATGGTCTCG GTGACGGCGC GGCGTCTC GCGGGGGCGC

FIGURE 21
(SHEET 3)

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9541 AGTTGGAAGA CGCCGCCCGT CATGTCCCGG TTATGGGTTG GCGGGGGCCT GCCATGCGGC
 9601 AGGGATAACGG CGCTAACGAT GCATCTCAAC AATTGTTGTC TAGGTAACCTCC GCGGCCGAGG
 9661 GACCTGAGCG AGTCCGCATC GACCGGATCG GAAAACCTCT CGAGAAAGGC GTCTAACCAAG
 9721 TCACAGTCGC AAGGTAGGCT GAGCACCCTG CGGGGCCGCA GCGGGCGGCG GTCGGGGTG
 9781 TTTCTGGCGG AGGTGCTGCT GATGATGTA TTAAAGTAGG CGGTCTTGAG ACGGCGGATG
 9841 GTCGACAGAAA GCACCATGTC CTGGGTCCG GCCTGCTGAA TGCGCAGGCG GTCGGCCATG
 9901 CCCCAGGCTT CGTTTGACA TCGGCGCAGG TCTTTGAGT AGTCCTGCA GAGCCTTCT
 9961 ACCGGGACTT CTTCTCTCC TTCTCTTGT CCTGCATCTC TTGCATCTAT CGCTGCGGCG
 10021 GCGGCCGAGT TTGGCCGTAG GTGGCGCCCT CTTCTCCCCA TGCGTGTGAC CCCGAAGCCC
 10081 CTCATCGGCT GAAGCAGGGC TAGGTGCGGCG ACAACCGCCT CGGCTAAATAT GGCGCTGCTG
 10141 ACCTGCGTGA GGGTAGACTG GAAGTCATCC ATGTCACAA AGCGGTGGTA TGCGCCCGTG
 10201 TTGATGGGT AAGTGCAGTT GGCCTAAACG GACCAAGTTAA CGGTCTGGTG ACCGGCTG
 10261 GAGAGCTCGG TGTACCTGAG ACAGCAGTAA GCGCTCGAGT CAAATACGTA GTCGTTGCAA
 10321 GTCCGCACCA GGTACTGGTA TCCCACCAAA AAGTGGCGGCG GCGGCTGGCG GTAGAGGGC
 10381 CAGCGTAGGG TGGCGGGGCG TCCGGGGGCG AGATCTTCCA ACATAAGGCG ATGATATCCG
 10441 TAGATGTACC TGGACATCCA GGTGATGCCG CGGGCGGTGG TGGAGGCGCG CGGAAAGTCG
 10501 CGGACGCGGT TCCAGATGTT GCGCAGCGGC AAAAGTGCT CCATGGTCGG GACGCTCTGG
 10561 CCGGTCAGGC GCGCGCAATC GTGACGCTC TAGACCGTGC AAAAGGAGAG CCTGTAAGCG
 10621 GGCACCTTC CGTGGTCTGG TGGATAAATT CGCAAGGTTA TCATGGCGGA CGACCGGGGT
 10681 TCGAGCCCCG TATCCGGCCG TCCGCGTGA TCCATGCCTG TACCGCCCGC GTGTCGAACC
 10741 CAGGTGTGCG ACAGTCAGACA ACAGGGGGAGT GCTCTTTTG GCTCCCTTCC AGGCGCGGCG
 10801 GCTGCTGCGC TAGCTTTTT GGCCACTGGC CGCGCGCAGC GTAAGCGGTT AGGCTGGAAA
 10861 GCGAAAGCAT TAAGTGGCTC GCTCCCTGTA GCCGGAGGGT TATTTCCAA GGGTTGAGTC
 10921 GCGGGACCCC CGGTTCGAGT CTGGACCGG CGGACTGCG GCGAACGGGG GTTTGCCTCC
 10981 CCGTCATGCA AGACCCCGCT TCGAAATTCC TCCGAAACA GGGACGAGCC CCTTTTTTGC
 11041 TTTTCCCAGA TGCATCCGGT GCTGCGGAG ATGCGCCCCC CTCTCAGCA GCGGCAAGAG
 11101 CAAGAGCAGC GGCAGACATG CAGGGCACCC TCCCCTCTC CTACCGCGTC AGGAGGGCG
 11161 ACATCCGGG TTGACCGGGC AGCAGATGGT GATTACGAA CCCCAGGGCG CGGGGCCCG
 11221 CACTACCTGG ACTTGGAGGA GGGCGAGGGC CTGGCGCGC TAGGAGCGCC CTCTCCTGAG
 11281 CGGTACCCAA GGGTGCAGCT GAAGCGTGT ACAGCGTGAG CGTACGTGCC GCGGCAGAAC
 11341 CTGTTTCGCG ACCCGCAGGG AGAGGAGCC GAGGAGATGC GGGATCGAAA GTTCCACGCA
 11401 GGGCGCGAGC TGCGGCATGG CCTGAATCGC GAGCGTTGC TGCGCGAGGA GGACTTTGAG
 11461 CCCGACGCGC GAACCGGGAT TAGTCCCGCG CGCGCACACG TGGCGCCCGC CGACCTGGTA
 11521 ACCGCATAACG AGCAGACGGT GAACCCAGGAG ATTAACCTTC AAAAAGCTT TAACAACAC
 11581 GTGCGTACGC TTGTGGCGC CGAGGAGGTG GCTATAGGAC TGATGCATCT GTGGGACTTT
 11641 GTAAGCGCGC TGGAGAAAA CCCAAATAGC AAGCCGCTCA TGCGCGAGCT GTTCCCTATA
 11701 GTGCGACACA GCAGGGACAA CGAGGCAITC AGGGATGCCG TGCTAAACAT AGTAGAGCCC
 11761 GAGGGCCGCT GGCTGCTCGA TTGATAAAC ATCCTGCAGA GCATAGTGGT GCAGGAGCGC
 11821 AGCTTGAGCC TGGCTGACAA GGTGGCGGC ATCAACTATT CCATGCTTAG CCTGGGCAAG
 11881 TTTTACGCC GCAAGATATA CCATACCCCT TACGTTCCA TAGACAAGGA GGTAAAGATC
 11941 GAGGGGTCT ACATGCGCAT GCGCTGAAAG TGCTTACCT TGAGCGACGA CCTGGGGCTT
 12001 TATCGCAACG AGCGCATCCA CAAGGCCGTG AGCGTGAGCC GCGGCCGCGA GCTCAGCGAC
 12061 CGCGAGCTGA TGACAGCCT GCAAAAGGGCC CTGGCTGGCA CGGGCAGCGG CGATAGAGAG
 12121 GCGGAGTCCT ACTTGTACGC GGGCGCTGAC CTGGCTGGG CCCCCAGGCC ACGCGCCCTG
 12181 GAGGGCAGCTG GGGCCGGACG TGGGCTGGCG GTGGCACCCCG CGCGCGCTGG CAACGTCGGC
 12241 GCGCTGGAGG AATATGACGA GGACGATGAG TACGAGCCAG AGGACGGCGA GTACTAAGCG
 12301 GTGATGTTT TGATCAGATG ATGCAAGACG CAACGGACCC GGGGGTGCAG GCGGCCCTGC
 12361 AGAGCCAGGC GTCCGGCCTT AACTCCACGG ACGACTGGCG CCAGGTATG GACCGCATCA
 12421 TGTCCCTGAC TGCGCGCAAT CCTGACGCGT TCCGGCAGCA GCCGCGAGGC AACCGGCTCT
 12481 CGCGAATTCT GGAAGCGGTG GTCCCGCGC GCGCAAACCC CACGCACGAG AAGGTGCTGG
 12541 CGATCGTAA CGCGCTGGCC GAAAACAGGG CCATCCGGCC CGACGAGGCC GGCGCTGGTCT
 12601 ACGACGCGCT GCTTCAGCGC GTGGCTCGTT ACAACAGCGG CAACGTGAG ACCAACCTGG
 12661 ACCGGCTGGT GGGGGATGTG CGCGAGGCCG TGGCGCAGCG TGAGCGCGCG CAGCAGCAGG
 12721 GCAACCTGGG CTCCATGGTT GCACTAAACG CCTTCTGAG TACACAGCCC GCCAACGTGC
 12781 CGCGGGGACA GGAGGACTAC ACCAACCTTG TGAGCCACT GCGGCTAATG GTGACTGAGA
 12841 CACCGCAAAG TGAGGTGTAC CAGTCTGGGC CAGACTATT TTTCCAGACC AGTAGACAAG
 12901 GCCTGCAGAC CGTAAACCTG AGCCAGGCTT TCAAAACCTT GCAGGGCTG TGGGGGTGC

FIGURE 21
(SHEET 4)

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12961 GGGCTCCCAC AGGCAGCCGC GCGACCGTGT CTAGCTTGCT GACGCCAAC TCGGCCCTGT
 13021 TGCTGCTGCT AATAGCGCCC TTCACGGACA GTGGCAGCGT GTCCCGGGAC ACATACTTAG
 13081 GTCACTTGCT GACACTGTAC CGCGAGGCCA TAGGTCAAGGC GCATGTGGAC GAGCATACTT
 13141 TCCAGGAGAT TACAAGTGTAC AGCCGCGCAGC TGGGGCAGGA GGACACGGGC AGCCTGGAGG
 13201 CAACCCCTAAA CTACCTGCTG ACCAACCGGC GGCAGAAGAT CCCCTCGITG CACAGTTAA
 13261 ACAGCGAGGA GGAGCGCATT TTGCGCTACG TGCAGCAGAG CGTGAGCCCT AACCTGATGC
 13321 GCGACGGGGT AACGCCAGC GTGGCGCTGG ACATGACCGC GCGCAACATG GAACCGGGCA
 13381 TGTATGCCTC AAACCGGCCG TTATCAACC GCCTAATGGGA CTACTTGCT CGCGCGGGCG
 13441 CGGTGAACCC CGAGTATTC ACCAATGCCA TCTTGAACCC GCACGGCTA CCGCCCCCTG
 13501 GTTCTACAC CGGGGGATTG GAGGTGCGCC AGGGTAACGA TGAGTTCTC TGGGACGACA
 13561 TAGACGACAG CGTGTTC CCGCAACCGC AGACCCCTGCT AGAGTTGCAA CAGCGCGAGC
 13621 AGGCAGAGGC GGCGCTGCGA AAGGAAAGCT TCCGCAGGCC AAGCAGCTG TCCGATCTAG
 13681 GCGCTGCGC CCGCGGTCA GATGCTAGTA GCCCATTCC AAGCTTGATA GGGTCTCTTA
 13741 CCAGCACTCG CACCACCCGC CGCGCGCTGC TGGGCGAGGA GGAGTACCTA AACAACTCGC
 13801 TGCTGCAGCC GCAGCGCGAA AAAAACCTGC CTCCGGCATT TCCAACAAC GGGATAGAGA
 13861 GCCTAGTGGAA CAAGATGAGT AGATGGAAGA CGTACCGCGA GGAGCACAGG GACGTGCCAG
 13921 GCCCGGCC GCCCCCGT CGTCAAAGGC ACGACCGTCA GCGGGGTCTG GTGTGGGAGG
 13981 AGATGACTC GGCAGACGAC AGCAGCGTCC TGGATTGGG AGGGAGTGGC AACCCGTTG
 14041 CGCACCTTCG CCCCAGGCTG GGGAGAATGT TTAAAAAAA AAAAAGCATG ATGAAAATA
 14101 AAAACTCAC CAAGGCCATG GCACCGAGCG TTGGTTTCT TGTATTCCCC TTAGTATGCG
 14161 GCGCGCGGGCG ATGTATGAGG AAGGTCCTCC TCCCTCTAC GAGAGTGTGG TGAGCGGGC
 14221 GCCAGTGGCG GCGGGCTGG GTTCTCCCTT CGATGCTCCC CTGGACCCGC CGTTTGTGCC
 14281 TCCCGGGTAC CTGCGGCTA CCGGGGGGAG AAACAGCATC CGTTACTCTG AGTTGGCACC
 14341 CCTATTCGAC ACCACCCGTG TGTACCTGGT GGACAACAAG TCAACGGATG TGGCATCCCT
 14401 GAACTACCAG AACGACCACA GCAACTTCT GACCACGGTC ATTCAAAACA ATGACTACAG
 14461 CCCGGGGGAG GCAAGCACAC AGACCATCAA TCTTGACGAC CGTTCGCACT GGGCGGGCGA
 14521 CCTGAAACCC ATCCTGCATA CCAACATGCC AAATGTGAAC GAGTTCATGT TTACCAATAA
 14581 GTTTAAGGCG CGGGTGTGG TGTCGCGCTT GCCTACTAAG GACAATCAGG TGGAGCTGAA
 14641 ATACGAGTGG GTGGAGTTCA CGCTGCCGA GGGCAACTAC TCCGAGACCA TGACCATAGA
 14701 CCTTATGAAC AACGGATCG TGGAGCACTA CTTGAAAGTG GGCAGACAGA ACGGGGTTCT
 14761 GGAAAGCGAC ATCGGGTAA AGTTTGACAC CCGCAACTTC AGACTGGGGT TTGACCCCGT
 14821 CACTGGTCTT GTCATGCCGT GGGTATATAAC AAACGAAGCC TTCCATCCAG ACATCATTIT
 14881 GCTGCCAGGA TCGGGGGTGG ACTTCACCCA CAGCCGCCTG ACAACTTGT TGGCATCCG
 14941 CAAGCGGCAA CCCTTCCAGG AGGGCTTTAG GATCACCTAC GATGATCTGG AGGGTGGTAA
 15001 CATTCCCGCA CTGTTGGATG TGGACGCCCTA CCAGGGGAG TTGAAAGATG ACACCGAACCA
 15061 GGGCGGGGGT GGCGCAGGCC GCAGCAACAG CAGTGGCAGC GGGCGGGAG AGAACTCCAA
 15121 CGCGGCAGCC GCGGAATGC AGCGGGTGGG GGACATGAAC GATCATGCCA TTCGCGCGA
 15181 CACCTTGC ACACGGCTG AGGAGAAGCG CGCTGAGGCC GAAGCAGCGG CGGAAGCTGC
 15241 CGCCCCCGCT GCGCAACCCG AGGTCGAGAA GCCTCAGAAG AAACCGGTGA TCAAACCCCT
 15301 GACAGAGGAC AGCAAGAAC GCAGTTACAA CCTAATAAGC AATGACAGCA CCTTCACCCA
 15361 GTACCGCAGC TGGTACCTTG CATACAACTA CGCGACCCCT CAGACCGGAA TCCGCTCATG
 15421 GACCCCTGCTT TGCACCTCTG ACGTAACCTG CCGCTGGAG CAGGTCTACT GGTGTTGCC
 15481 AGACATGATG CAAGACCCCG TGACCTTCCG CTCCACGCG CAGATCAGCA ACTTTCCGGT
 15541 GGTGGGCGCC GAGCTGTTGC CGTGCACCTC CAAGAGCTTCA TACAACGACC AGGCCGTCTA
 15601 CTCCCAACT ATCCGCCAGT TTACCTCTT GACCCACGT TICAATCGCT TTCCCGAGAA
 15661 CCAGATTTTG GCGGCCCGC CAGCCCCCAC CATCACCAAC GTCAGTGAAA ACGTTCTGC
 15721 TCTCACAGAT CACGGGACGC TACCGCTGCC CAACAGCATE GGAGGGAGTCC AGCGAGTGAC
 15781 CATTACTGAC GCCAGACGCC GCACCTGCCCT CTACGTTAC AAGGCCCTGG GCATAGCTC
 15841 GCCGCGCGTC CTATCGAGCC GCACCTTTTG AGCAAGCATG TCCATCCCTA TATCGCCAG
 15901 CAATAACACA GGCTGGGGCC TGCGCTTCCC AAGCAAGATG TTGGCGGGG CCAAGAACG
 15961 CTCCGACCAA CACCCAGTGC GCGTGCAGCG GCACTACCGC GCGCCCTGG GCGCGCACAA
 16021 ACGGGGCCGC ACTGGGCGCA CCACCGTCGA TGACGCCATC GACGCCGTGG TGGAGGAGGC
 16081 GCGCAACTAC ACGCCCACGC CGCCACCAAGT GTCCACAGTG GACGCCGGCCA TTCAGACCGT
 16141 GGTGGCGGGA GCCCGCGCT ATGCTAAAT GAAGAGACGG CGGAGGCGCG TAGCACGTCG
 16201 CCACCGCCGC CGACCCGGCA CTGCGGCCA ACGCGCGCG GCGGCCCTGC TTAACCGCGC
 16261 ACGTGCGCACCGGGGGGG CGGCCATGGG GGCGCTCGA AGGCTGGCG CGGGTATTGT
 16321 CACTGTGCCCTT CCCAGGTCGA GGCGACGAGC GGCGCCCGCA GCAGCGCGG CCATTAGTGC

FIGURE 21
(SHEET 5)

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16381 TATGACTCAG GGTCGCAGGG GCAACGTGTA TTGGGTGCGC GACTCGGTTA GCGGCCCTGCG
 16441 CGTGCCCGTG CGCACCCGCC CCCCCGCGCAA CTAGATTGCA AGAAAAAAACT ACTTAGACTC
 16501 GTACTGTTGT ATGTATCCAG CGGCGGCGGC GCGCAACGAA GCTATGTCCA AGCGCAAAAT
 16561 CAAAGAACAG ATGCTCCAGG TCATCGCGCC GGAGATCTAT GGCCCCCGA AGAAGGAAGA
 16621 GCAGGATTAC AAGCCCCGAA AGCTAAAGCG GGTCAAAAAG AAAAGAAAAG ATGATGATGA
 16681 TGAACCTTGAC GACGAGGTGG AACTGCTGCA CGCTACCGCG CCCAGGCAC ACCGTACAGTG
 16741 GAAAGGTGCA CGCGTAAAAG GTGTTTGC CGCCGGCACC ACCGTAGTCT TTACGCCCG
 16801 TGAGCGCTCC ACCCGCACCT ACAAGGGCGT GTATGATGAG GTGTAACGGCG ACGAGGACCT
 16861 GCTTGAGCAG GCCAACGAGC GCCTCGGGGA GTTGCCTAC GGAAAGCGGC ATAAGGACAT
 16921 GCTGGCGTGT CGCGCTGGACG AGGGCAACCC AACACCTAGC CTAAGGCCCG TAACACTGCA
 16981 GCAGGTGCTG CCCCGCGCTTG CACCGTCCGA AGAAAAGCGC GGCTAAAGC GCGAGTCTGG
 17041 TGACTTGCA CCCACCGTGC AGCTGATGGT ACCCAAGCGC CAGCGACTGG AAGATGCTT
 17101 GGAAAAAAATG ACCGTGGAAC CTGGGCTGGA GCGCGAGGTC CGCGTGCAGC CAATCAAGCA
 17161 GGTGGCGCCG GGACTGGGCG TGCAGACCGT GGACGTTAGC ATACCCACTA CCAGTAGCAC
 17221 CAGTATTGCC ACCGCCACAG AGGGCATGGA GACACAAAAGC TCCCCGGTTG CCTCAGCGGT
 17281 GGCGGATGCC GCGGTGCAAG CGGTGCGCTGC GGCGCGCTCC AAGACCTCTA CGGAGGTGCA
 17341 AACGGACCCG TGGATGTTTC GCGTTTCAGC CCCCCGGCGC CGCGCGGTT CGAGGAAGTA
 17401 CGGCGCCGCC AGCGCGCTAC TGCCCAGATA TGCCCTACAT CCTTCCATTG CGCCTACCCC
 17461 CGGCTATCGT GGCTACACCT ACCGCCCCAG AAGACGAGCA ACTACCCGAC GCGAACAC
 17521 CACTGGAACCG CGCCGCCGCC GTGCCCGTGC CCAGCCCGTG CTGGCCCGA TTTCCGTGCG
 17581 CAGGGTGGCT CGCGAAGGAG CGAGGACCCG GGTGCTGCCA ACAGCGCGCT ACCACCCAG
 17641 CATCGTTAA AAGCCGGCTC TTGTTGGCTC TGCAAGATATG GCCCTCACCT GCGCCCTCCG
 17701 TTTCGGGTG CGGGATTCC GAGGAAGAAAT GCACCGTAGG AGGGGCATGG CGGGCCACGG
 17761 CCTGACGGGC GGCATGCGTC GTGCGCACCA CGCGCGCGG CGCGCGTCGC ACCGTCGCA
 17821 GCGCGGGGT ATCCTGCCCT CCCTTATTCC ACTGATGCC GCGCGGATTG GCGCCGTGCG
 17881 CGGAATTGCA TCCGTGGCCT TGCAGGCGCA GAGACACTGA TAAAAAAACAA GTTGCATGTG
 17941 GAAAAATCAA AATAAAAAGT CTGGACTCTC ACGCTCGCTT GGTCTGTAA CTATTTGTAA
 18001 GAATGGAAAGA CATCAACTTT GCGTCTCTGG CCCCCGGACCA CGGCTCGCGC CGGTTCATGG
 18061 GAAACTGGCA AGATATCGC ACCAGCAATA TGAGCGGTGG CGCCCTTCAGC TGGGGCTCGC
 18121 TGTGGAGCGG CATTAAAAAT TCGGTTCCA CCGTTAAGAA CTATGGCAGC AAGGCCTGGA
 18181 ACAGCAGCAC AGGGCAGATG CTGAGGGATA AGTTGAAAGA GCAAAATTTCAACAAAAGG
 18241 TGGTAGATGG CCTGGCTCT GGCATTAGCG GGGTGGTGG CCTGGCCAAAC CAGGCAGTGC
 18301 AAAATAAGAT TAACAGTAAG TTGATCCCC GCGCCCTTCAGT AGAGGAGGCT CCACCGGCCG
 18361 TGGAGACAGT GTCTCCAGAG GGGCTGGCG AAAAGCGTCC CGGCCCGGAC AGGGAAAGAAA
 18421 CTCTGGTGAC GCAAATAGAC GAGCCTCCCT CGTACGGAGA GGCACTAAAG CAAGGCTGCA
 18481 CCACCAACCG TCCCAGCGC CCCATGGCTA CGGGAGTGT GGGCCAGCAC ACACCCGTA
 18541 CGCTGGACCT GCCTCCCCCCC GCGACACCC AGCAGAAACC TGTCGTCGCC GGCCCCGACCG
 18601 CGTTGTTGT AACCCGTCCT AGCCGCGCGT CCTGCGCCG CGCCGCCAGC GGTCCCGAT
 18661 CGTTGCGGCC CGTAGCCAGT GGCAACTGGC AAAGCACACT GAAACAGCATC GTGGGTCTGG
 18721 GGGTGCATTC CCTGAAGCGC CGACGATGCT TCTGAATAGC TAACGTGTCG TATGTGTGTC
 18781 ATGTATGCCG CCTATGTCGCC GCCAGAGGAG CTGCTGAGCC GCGCGCGGCC CGCTTTCCAA
 18841 GATGGCTACC CCTTCGATGA TGCCGCACTG GTCTTACATG CACATCTCGG GCCAGGACGC
 18901 CTCGGAGTAC CTGAGCCCCG GGCTGGTGC GTTTGGCCGC GCCACCGAGA CGTACTTCAG
 18961 CCTGAATAAC AAGTTAGAA ACCCCACGGT GGCGCTCTACG CACGACGTGA CCACAGACCG
 19021 GTCCCAGCGT TTGACGCTGC GGTTCATCCC TGTGGACCGT GAGGATACTG CGTACTCGTA
 19081 CAAGGGCGGG TTCACCCCTAG CTGTGGGTGA TAACCGTGTG CTGGACATGG CTTCCACGTA
 19141 CTTTGACATC CGCGCGTGC TGGACAGGGG CCCTACTTTT AAGCCCTACT CTGGCACTGC
 19201 CTACAACGCC CTGGCTCCCA AGGGTGGCCC AAATCCTTGC GAATGGGATG AAGCTGCTAC
 19261 TGCTCTGAA ATAAACCTAG AAGAACAGGA CGATGACAAC GAAGACGAAG TAGACGAGCA
 19321 AGCTGAGCAG CAAAAAAACTC ACGTATTTGG CGAGGCGCT TATTCTGGTA TAAATATTAC
 19381 AAAGGGAGGT ATTCAAATAG GTGTCGAAGG TCAAACACCT AAATATGCCG ATAAAACATT
 19441 TCAACCTGAA CCTCAAATAG GAGAATCTCA GTGGTACGAA ACTGAAATTG ATCATGAGC
 19501 TGGGAGAGTC CTTAAAAAGA CTACCCCAAT GAAACCATGT TACGGTTCAT ATGCAAAC
 19561 CACAAATGAA AATGGAGGGC AAGGCATTCT TGAAAGCAA CAAAATGGAA AGCTAGAAAG
 19621 TCAAGTGGAA ATGCAATTTC TCTCAACTAC TGAGGGCACC GCAGGCAATG GTGATAACTT
 19681 GACTCCTAAA GTGGTATTGT ACAGTGAAGA TGTAGATATA GAAACCCCGAG AACTCATAT
 19741 TTCTTACATG CCCACTATTA AGGAAGGTAA CTCACCGAGAA CTAATGGGCC AACAACTAT

19801 GCCAACAGG CCTAATTACA TTGCTTTAG GGACAATTTT ATTGGTCTAA TGTATTACAA
 19861 CAGCACGGGT AATATGGGT TTCTGGCGGG CCAAGCATCG CAGTTGAATG CTGTTGTAGA
 19921 TTTGCAAGAC AGAACACAG AGCTTCATA CCAGCTTTG CTTGATTCCA TTGGTGATAG
 19981 AACCAAGTAC TTTTCTATGT GGAATCAGGC TGTTGACAGC TATGATCCAG ATGTTAGAAT
 20041 TATTGAAAAT CATGGAACGTG AAGATGAACT TCCAAATTAC TGCTTTCCAC TGGGAGGTGT
 20101 GATTAATTACA GAGACTCTTA CCAAGGTAAA ACCTAAAACA GGTCAGGAAA ATGGATGGGA
 20161 AAAAGATGCT ACAGAATTTC CAGATAAAA TGAAAATAGA GTTGGAAATA ATTTGCCAT
 20221 GGAAATCAAT CTAAATGCCA ACCTGTGGAG AAATTTCTG TACTCCAACA TAGCGCTGTA
 20281 TTTGCCGAC AAGCTAAAGT ACAGTCCTTC CAACGTTAAA ATTTCTGATA ACCCAAACAC
 20341 CTACGACTAC ATGAACAAGC GAGTGGTGGC TCCCCGGTTA GTGGACTGCT ACATTAACCT
 20401 TGGAGCACGC TGGTCCCTTG ACTATATGGA CAACGTCAAC CCATTTAACCC ACCACCGCAA
 20461 TGCTGGCCTG CGCTACCGCT CAATGTTGCT GGGCAATGGT CGCTATGTGC CCTTCCACAT
 20521 CCAGGTGCCT CAGAAGTTCT TTGCCATTAA AAACCTCCCTT CTCCCTGCCGG GCTCATACAC
 20581 CTACGAGTGG AACTTCAGGA AGGATGTTAA CATGGTTCTG CAGAGCTCCC TAGGAAATGA
 20641 CCTAAGGGTT GACGGAGCCA GCATTAAGTT TGATAGCATT TGCTTTACCG CCACCTCTT
 20701 CCCCCATGGCC CACAACACCG CCTCCACGCT TGAGGCCATG CTTAGAAACG ACACCAACGA
 20761 CCAGTCCCTT AACGACTATC TCTCCGCCGC CAACATGCTC TACCCCTATAC CCGCCAACGC
 20821 TACCAACGTG CCCATATCCA TCCCCCTCCCG CAACTGGCGC GCTTTCCCGG GCTGGGCCCTT
 20881 CACGCGCCTT AAGACTAAGG AAACCCCCATC ACTGGGCTCG GGCTACGACC CTTATTACAC
 20941 CTACTCTGGC TCTATACCCCT ACCTAGATGG AACCTTTAC CTCAACCACA CCTTTAAGAA
 21001 GGTGGCCATT ACCTTTGACT CTTCCTGTCAG CTGGGCTGGC AATGACCGCC TGCTTACCCC
 21061 CAACGAGTTT GAAATTAAGC GCTCAGTTGA CGGGGAGGGT TACAACGTTG CCCAGTGTAA
 21121 CATGACCAAA GACTGGTTC TGTTACAAAT GCTAGCTAAC TACAACATTG GCTACCAAGGG
 21181 CTTCTATATC CCAGAGAGCT ACAAGGACCG CATGTAATCC TTCTTTAGAA ACTTCCAGGC
 21241 CATGAGCCGT CAGGTGGTGG ATGATACTAA ATACAAGGAC TACCAACAGG TGGGCATCCT
 21301 ACACCAACAC AACAACTCTG GATTTGTTGG CTACCTTGCC CCCACCATGC GCGAAGGACA
 21361 GGCCTACCCCT GCTAACCTCC CCTATCCGCT TATAGGCAAG ACCGAGTTG ACAGCATTAC
 21421 CCAGAAAAAG TTTCTTGCG ATCGCACCC TTGGCGCATC CCATTCTCCA GTAACTTTAT
 21481 GTCCATGGGC GCACATCACAG ACCTGGGCCA AAACCTTCTC TACGCCAACT CCGCCCCACGC
 21541 GCTAGACATG ACTTTTGAGG TGGATCCCCAT GGACGAGCCC ACCCTTCTTT ATGTTTTGTT
 21601 TGAAGTCTTT GACGGGGTCC GTGTGCACCC GCGCACCAGC GGCGTCTACG AAACCGTGTAA
 21661 CCTGCGCACG CCCCCCTCGG CGGGCAACGC CACAACTAA AGAAGCAAGC AACATCAACA
 21721 ACAGCTGCCG CCATGGGCTC CAGTGAGCAG GAACTGAAAG CCATTGTCAA AGATCTTGGT
 21781 TGTGGGCCAT ATTTTTGGG CACCTATGAC AAGCGCTTTC CAGGCTTTGT TTCTCCACAC
 21841 AAGCTCGCCT GCGCCATAGT CAATACGGCC GGTCCCGAGA CTGGGGCGT ACACTGGATG
 21901 GCCTTTGCCT GGAACCCGCA CTCAAAACAA TGCTACCTCT TTGAGCCCTT TGGCTTTCT
 21961 GACCAGCGAC TCAAGCAGGT TTACCACTT GAGTACGAGT CACTCTGCG CCGTAGCGCC
 22021 ATTGCTTCTT CCCCCGACCG CTGTATAACG CTGGAAAAGT CCACCCAAAG CGTACAGGGG
 22081 CCCAACTCGG CGGCCCTGTGG ACTATTCTGC TGCATGTTTC TCCACGCCCT TGCCAACCTGG
 22141 CCCCCAAACTC CCATGGATCA CAACCCCCACC ATGAACCTTA TTACCGGGGT ACCCAACACTCC
 22201 ATGCTCAACA GTCCCCAGGT ACAGCCCACC CTGCGTCGCA ACCAGGAACA GCTCTACAGC
 22261 TTCCCTGGAGC GCCACTCGCC CTACTTCCGC AGCCACAGTG CGCAGATTAG GAGGCCACT
 22321 TCTTTTGTC ACTTGAAAAA CATGAAAAAA TAATGACTA GAGACACTTT CAATAAAAGGC
 22381 AAATGCTTTT ATTTGTACAC TCTCGGGTGA TTATTTACCC CCACCCCTTGC CGTCTCGGCC
 22441 GTTTAAAAAT CAAAGGGTT CTGCCGCGCA TCGCTATGCG CCACTGGCAG GGACACGTTG
 22501 CGATACTGGT GTTGTAGTGT CCACCTAAAC TCAGGCACAA CCATCCCGGG CAGCTCGGTG
 22561 AAGTTTCAC TCCACAGGGCT GCGCACCATC ACCAACCGGT TTAGCAGGTC GGGCGCCGAT
 22621 ATCTTGAACT CGCAGTTGGG GCCTCCGCCCC TGCGCGCGCG AGTTGCGATA CACAGGGTTG
 22681 CAGCACTGGA ACACATATCAG CGCCGGGTGG TGCACGCTGG CCAGCACGCT CTTGTCGGAG
 22741 ATCAGATCCG CGTCCAGGT CTCCCGCTTG CTCAGGGCGA ACGGAGTCAA CTTTGGTAC
 22801 TGCCCTCCCA AAAAGGGCGC GTGCCCGAGC TTGAGTTGC ACTCGCACCG TAGTGGCATC
 22861 AAAAGGTGAC CGTGGCCGGT CTGGCGTTA GGATACAGCG CCTGCATAAA AGCCTTGTATC
 22921 TGCTTAAAG CCACCTGAGC CTTTGGCGTC TCAGAGAAGA ACATGCCGCA AGACTTGGCG
 22981 GAAAATGAT TGGCGGGACA GGCGCGCTCG TGCAACGAGC ACCTTGCCTC GGTGTTGGAG
 23041 ATCTGCACCA CATTGCGCC CCACCGGTT TTCACGATCT TGGCTTGCT AGACTGCTCC
 23101 TTCAGCGCGC GCTGCCCCGT TTCGCTCGTC ACATCCATT CAATCACGTG CTCCATTATTT
 23161 ATCATAATGC TTCCGTGTAG ACACCTAAAGC TCGCCCTCGA TCTCAGCGCA GCGGTGCGAGC

23221 CACAACGCGC AGCCC GTGGG CTCGTGATGC TTGTAGGTCA CCTCTGCAA CGACTGCAGG
 23281 TACGCCCTGCA GGAATCGCCC CATCATCGTC ACAAAAGGTCT TGTTGCTGGT GAAGGTCAGC
 23341 TGCAACCCGC GGTGCTCCCTC GTTCAGCCAG GTCTTGATA CGGCCGCCAG AGCTTCCACT
 23401 TGGTCAGGCA GTAGTTGAA GTTCGCCCTT AGATCGTTAT CCACGTGGTA CTITGTCATC
 23461 AGCGCGCGCG CAGCCCTCCAT GCCCTTCTCC CACGCAGACA CGATCGGCAC ACTCAGCGG
 23521 TTCATCACCG TAATTTCACT TTCCGCTTCG CTGGGCTCTT CCTCTTCCTC TTGCGTCCGC
 23581 ATACCACGCG CCACTGGGTC GTCTTCATTC AGCCGGCGCA CTGTGCGCTT ACCTCCCTTG
 23641 CCATGCTGTA TTAGCACCGG TGGGTTGCTG AAACCCACCA TTITGAGCGC CACATCTTCT
 23701 CTTTCTTCCT CGCTGTCAC GATTACCTCT GGTGATGGCG GGCGCTCGGG CTTGGGAGAA
 23761 GGGCGCTCTC TTTTCTTCCTT GGGCGCAATG GCCAAATCCG CCGCCGAGGT CGATGGCCGC
 23821 GGGCTGGGTG TGCGCGGCAC CAGCGCGTCT TGTGATGAGT CTTCTCGTC CTGGGACTCG
 23881 ATACGCCGCC TCATCCGCTT TTTTGGGGC GCCCCGGGGAG GCGGGCGCGA CGGGGACGGG
 23941 GACGACACGT CCTCCATGGT TGGGGGACGT CGCGCCGCAC CGCGTCCGCG CTCGGGGTG
 24001 GTTTCGCGCT GCTCCTCTTC CCGACTGGGC ATTTCCTTCT CCTATAGGCA GAAAAAGATC
 24061 ATGGAGTCAG TCGAGAAGAA GGACAGCTA ACCGCCCCCT CTGAGTTCGC CACCACGCC
 24121 TCCACCGATG CGGCCAACGC GCCTACCAAC TTCCCCGTCG AGGCACCCCCC GCTTGAGGAG
 24181 GAGGAAGTGA TTATCGAGCA GGACCCAGGT TTGTAAGCG AAGACGACGA GGACCGCTCA
 24241 GTACCAACAG AGGATAAAAA GCAAGACCGAG GACAACGCAG AGGCAAACGA GGAACAAGTC
 24301 GGGCGGGGGG ACGAAAGGCA TGGCGACTAC CTAGATGTGG GAGACGACGT GCTGTTGAAG
 24361 CATCTGCAGC GCCAGTGCAC CATTATCTGC GACGCGTTGC AAGAGCGCAG CGATGTGCC
 24421 CTCGCCATAG CGGATGTCAG CCTTGCCCTAC GAACGCCACC TATTCTCACC GCGCGTACCC
 24481 CCCAAACGCC AAGAAAACGG CACATGCGAG CCCAACCCGC GCCTCAACTT CTACCCGTA
 24541 TTTGCCGTGC CAGAGGTGCT TGCCACCTAT CACATCTTT TCCAAAATG CAAGATACCC
 24601 CTATCCTGCC GTGCCAACCG CAGCCGAGCG GACAAGCAGC TGGCCTTGCG GCAGGGCGCT
 24661 GTCATACCTG ATATCGCTC GCTCAACGAA GTGCCAAAAA TCTTTGAGGG TCTTGGACGC
 24721 GACGAGAAGC GCGCGGCAA CGCTCTGCAA CAGGAAAACA GCGAAAATGA AAGTCACTCT
 24781 GGAGTGTGG TGGAACCTCGA GGGTGACAAC GCGCGCCTAG CGTACTAAA ACGCACATC
 24841 GAGGTCAACCC ACTTTGCCTA CCCGGCACTT AACCTACCCC CCAAGGTCAT GAGCACAGTC
 24901 ATGAGTGAAGC TGATCGTGCG CCGTGCAG CCCCCTGGAGA GGGATGCAA TTTGCAAGAA
 24961 CAAACAGAGG AGGGCCTACC CGCAGTTGGC GACGAGCAGC TAGCGCGCTG GCTTCAAACG
 25021 CGCGAGGCTG CGCACTTGGA GGAGCGACGC AAACATATGA TGGCCCGCAGT GCTCGTTACC
 25081 GTGGAGCTTG AGTGCATGCA GCGGTTCTT GCTGACCCCG AGATGCGAGC CAAGCTAGAG
 25141 GAAACATTCG ACTACACCTT TCGACAGGGC TACGTACCCC AGGCTGCAA GATCTCCAAC
 25201 GTGGAGCTCT GCAACCTGGT CTCCCTACCTT GGAATTTCG ACGAAAACCG CCTTGGCAA
 25261 AACGTGCTTC ATTCCACGCT CAAGGGCGAG GCGCGCCGCG ACTACGTCCG CGACTGCGTT
 25321 TACTTATTC TATGCTACAC CTGGCAGACG GCCATGGCG TTTGGCAGCA GTGCTTGGAG
 25381 GAGTGCACCC TCAAGGAGCT CGAGAAACTC CTAAGCAGGAA ACTTGAAGGA CCTATGGACG
 25441 GCCTTCACCG AGGCGCTCCGT GGCGCGCAC CTGGGGAGA TCATTTCCC CGAACGCCCTG
 25501 CTTAAAACCC TGCAACAGGG TCTGCCAGAC TTCACCAAGTC AAAGCATGTT GCAGAACTTT
 25561 AGGAACCTTA TCCTAGAGCG CTCAGGAATC TTGCCCCGCA CTCGCTGTGC ACTTCTTAGC
 25621 GACTTTGTGC CCATTAAGTA CGCGGAATC CCTCCGCCGC TTTGGGGCCA CTGCTACCTT
 25681 CTGCACTGAG CCAACTACCT TGCCCTACAC TCTGACATAA TGGAAAGACGT GAGCGGTGAC
 25741 GGTCTACTGG AGTGTCACTG TCGCTGCAAC CTATGCACCC CGCACCGCTC CCTGGTTTGC
 25801 AATTGCGAGC TGCTTAACGA AAGTCAAATT ATCGTACCT TTGAGCTGCA GGGTCCCTCG
 25861 CCTGACGAAA AGTCCGCGGC TCCGGGGTTC AACTCACTC CGGGGCTGTG GACGTCGGCT
 25921 TACCTTCGCA AATTGTACC TGAGGACTAC CACGCCACG AGATTAGGTT CTACGAAGAC
 25981 CAATCCGCC CGCCAAATGC GGAGCTTACG GCCTCGTCA TTACCCAGGG CCACATTCTT
 26041 GGCAATTGC AAGCCATCAA CAAAGCCCG CAAGAGTTTC TGCTACGAAA GGGACGGGG
 26101 GTTTACTTGG ACCCCCCAGTC CGGGGAGGAG CTCAACCCAA TCCCCCGCC CGCGCAGGCC
 26161 TATCAGCAGC AGCCGCGGGC CCTTGCTTCC CAGGATGGCA CCCAAAAAGA AGCTGCAGCT
 26221 GCCGCCGCC CCCACGGACG AGGAGGAATA CTGGGACAGT CAGGAGAGGG AGGTTTTGGA
 26281 CGAGGAGGAG GAGGACATGA TGGAAAGACTG GGAGAGCCTA GACGAGGAAG CTTCCGAGGT
 26341 CGAAGAGGTG TCAGACGAAA CACCGTCACC CTCGCTCGCA TTCCCCCTCGC CGGGGCCACT
 26401 GAAATCGGCA ACCGGTTCCA GCATGGTAC AACCTCCGCT CCTCAGGCGC CGGGGCCACT
 26461 GCCCCTTCGC CGACCCAAAC GTAGATGGGA CACCACTGGA ACCAGGGCCG GTAAGTCCAA
 26521 GCAGCGCGCG CGGTAGGCC AAGAGCAACA ACAGCGCCAA GGCTACCGCT CATGGCGCGG
 26581 GCACAAGAAC GCCATAGTTG CTTGCTTGCA AGACTGTGGG GGCAACATCT CCTTCGCCCG

26641 CCGTTTCTT CTCTACCATC ACGGCGTGGC CTTCCCCGT AACATCCTGC ATTACTACCG
 26701 TCATCTCTAC AGCCCATACT GCACCGGGGG CAGCGGCAGC GGCAGCAACA GCAGCGGCCA
 26761 CACAGAACGA AAGGCAGCG GATAGCAAGA CTCTGACAAA GCCCAAGAAA TCCACAGCGG
 26821 CGGCAGCAGC AGGAGGAGA CGCGTGCCTC TGGCGCCCAA CGAACCCGTA TCGACCCGCG
 26881 AGCTTAGAAA CAGGATTTC CCCACTCTGT ATGCTATATT TCAACAGAGC AGGGGCAAG
 26941 AACAAAGAGCT GAAAATAAA AACAGGTCTC TGCGATCCCT CACCCGCAGC TGCGCTGTATC
 27001 ACAAAAGCGA AGATCAGCTT CGGCGCACGC TGGAAAGACGC GGAGGCTCTC TTCAGTAAAT
 27061 ACTGCGCGCT GACTTTAAG GACTAGTTTC GCGCCCTTTC TCAAATTAA GCGGAAAC
 27121 TACGTCACTC CCAGCGGCCA CACCCGGCGC CAGCACCTGT CGTCAGCGCC ATTATGAGCA
 27181 AGGAAATTCC CACGCCCTAC ATGTGGAGTT ACCAGCCACA ATGGGACTT GCGGCTGGAG
 27241 CTGCCCAAGA CTACTCAACC CGAATAAACT ACATGAGCGC GGGACCCAC ATGATATCCC
 27301 GGGTCAACGG AATCCGCGCC CACCGAAACC GAATTCTCTT GGAACAGGGC GCTATTACCA
 27361 CCACACCTCG TAATAACCTT AATCCCCGTA GTTGGCCCGC TGCCCTGGTG TACCAGGAAA
 27421 GTCCCGCTCC CACCACTGTG GTACTTCCA GAGACGCCA GGCGAAGTT CAGATGACTA
 27481 ACTCAGGGGC GCAGCTGCG GCGGGCTTTC GTCACAGGGT GCGGTCGCCC GGGCAGGGTA
 27541 TAACTCACCT GACAATCAGA GGGCGAGGTA TTCAGCTCAA CGACGAGTCG GTGAGCTCCT
 27601 CGCTTGGTCT CCGTCCGGAC GGGACATTTC AGATCGGCGG CGCCGGCCGT CCTTCATTCA
 27661 CGCCTCGTCA GGCAATCCTA ACTCTGCAGA CCTCGTCCTC TGAGCCGCGC TCTGGAGGCA
 27721 TTGGAACCTCT GCAATTAAATT GAGGAGTTTG TGCCATCGGT CTACTTTAAC CCCTTCTCGG
 27781 GACCTCCCGG CCACTATCCG GATCAATTAA TTCTTAACCT TGACGCGGTA AAGGACTCGG
 27841 CGGACGGCTA CGACTGAATG TTAAGTGGAG AGGCAGAGCA ACTGCGCCTG AAACACTGG
 27901 TCCACTGTGCG CGGCCACAAG TGCTTTGCCG GCGACTCCGG TGAGTTTTGC TACTTTGAAT
 27961 TGCCCGAGGGA TCATATCGAG GGCCCCGGCGC ACGGGCTCCG GCTTACCGCC CAGGGAGAGC
 28021 TTGCCCCGTAG CCTGATTCCG GAGTTTACCC AGGCCTCCCT GCTAGTTGAG CGGGACAGGG
 28081 GACCCCTGTGT TCTCACTGTG ATTTGCAACT GTCCCTAACCT TGGATTACAT CAAGATCTT
 28141 GTTGCACATCT CTGTGCTGAG TATAATAAAAT ACAGAAATTAA AAATATACTG GGGCTCCTAT
 28201 CGCCATCCTG TAAACGCCAC CGTCTTCACC CGCCCAAGCA ACCAACGGCG AACCTTACCT
 28261 GGTACTTTTA ACATCTCTCC CTCTGTGATT TACAACAGTT TCAACCCAGA CGGAGTGAGT
 28321 CTACGAGAGA ACCTCTCCGA GCTCAGCTAC TCCATCAGAA AAAACACCAAC CCTCCTTAC
 28381 TGCCGGGAAC GTACGAGTGC GTCAACGGGC GCTGCAACAC ACCTACCGCC TGACCGTTAA
 28441 CCAGACTTTT TCCGGACAGA CCTCAATAAC TCTGTTTACCA AGAACAGGAG GTGAGCTTAG
 28501 AAAACCTTA GGGTATTAGG CCAAAGGGC AGCTACTGTG GGGTTTATGA ACAATTCAAG
 28561 CAACTCTTACG GGCTATTCTA ATTCAAGTTT CTCTAGAATC GGGGTTGGGG TTATTCTCTG
 28621 TCTTGTGATT CTCTTTATTCT TTATACTAAC GCTTCTCTGC CTAAGGCTCG CGGCCTGCTG
 28681 TGTGCACATT TGCAATTAAATT GTCAGCTTTT TAAACGCTGG GGTGCGCCACC CAAGATGATT
 28741 AGGTACATAA TCCTAGGTTT ACTCACCCCTT GCGTCAGCCC ACGGTACCAAC CCAAAAGGTG
 28801 GATTITAAGG AGCAGCCTG TAAATGTTACA TTGCGAGCTG AAGCTAATGA GTGCACCACT
 28861 CTTATAAAAT GCACCCACAGA ACATGAAAAG CTGCTTATTG GCCACAAAAAA CAAAATGGC
 28921 AAGTATGCTG TTTATGCTAT TTGGCAGCCA GGTGACACTA CAGAGTATAA TGTTACAGTT
 28981 TTCCAGGGTA AAAGTCATAA AACTTTTATG TATACTTTTC CATTTATGA AATGTGCGAC
 29041 ATTACCATGT ACATGAGCAA ACAGTATAAG TTGTGGCCCC CACAAAATTG TGTGGAAAAC
 29101 ACTGGCACTT TCTGCTGCAC TGCTATGCTA ATTACAGTGC TCGTTTGGT CTGTACCCCA
 29161 CTCTATATTA AATACAAAAG CAGACGCAGC TTTATTGAGG AAAAGAAAAAT GCCTTAATT
 29221 ACTAAGGTAC AAAGCTAATG TCACCACTAA CTGCTTTACT CGCTGCTTGC AAAACAAATT
 29281 CAAAAAGTTA GCATTATAAT TAGAATAGGA TTTAAACCCC CGGGTCATTI CCTGCTCAAT
 29341 ACCATTCCCC TGAAACAATTG ACTCTATGTG GGATATGCTC CAGCGCTACA ACCITGAAGT
 29401 CAGGCTTCCT GGATGTCAGC ATCTGACTTT GGCCAGCACC TGTCGGCGGG ATTGTTCCA
 29461 GTCCAACCTAC AGCAGCCAC CCTAACAGAG ATGACCAACA CAACCAACGC GGCCGCCGCT
 29521 ACCGGACTTA CATCTACCAC AAATACACCC CAAGTTCTG CCTTTGTCAA TAACGGGAT
 29581 AACTGGGCA TGTGGTGGTT CTCCATAGCG CTTATGTTTG TATGCTTAT TATTATGTTGG
 29641 CTCATCTGCT GCCTAAAGCG CAAACCGGCC CGACCAACCA TCTATAGTCC CATCATTGTG
 29701 CTACACCCAA ACAATGATGG AATCCATAGA TTGGACGGAC TGAAACACAT GTTCTTTCT
 29761 CTTACAGTAT GATTAATGA GACATGATTC CTCGAGTTT TATATTACTG ACCCTTGTG
 29821 CGCTTTTTTG TGGGTGCTCC ACATTGGCTG CGGTTCTCA CATCGAAGTA GACTGCAATT
 29881 CAGCCTTCAC AGTCTATTG CTTCACGGAT TTGTCACCCCT CACGCTCATC TGCAGCCTCA
 29941 TCACTGTGGT CATCCGCTTT ATCCAGTGCA TTGACTGGGT CTGTGTGCGC TTTGCATATC
 30001 TCAGACACCCA TCCCCAGTAC AGGGACAGGA CTATAGCTGA GCTTCTTAGA ATTCTTTAAT

FIGURE 21
(SHEET 9)

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30061 TATGAAATT ACTGTGACTT TTCTGCTGAT TATTCGCACC CTATCTGCCTT TTTGGTCCCC
 30121 GACCTCCAAG CCTCAAAGAC ATATATCATG CAGATTCACT CGTATATGGA ATATTCCAAG
 30181 TTGCTACAAT GAAAAAAGCG ATCTTCCGA AGCCTGGTTA TATGCAATCA TCTCTGTTAT
 30241 GGTGTTCTGC AGTACCATCT TAGCCCTAGC TATATATCCC TACCTTGACA TTGGCTGGAA
 30301 ACGAATAGAT GCCATGAACC ACCCAACTTT CCCCGCGCCC GCTATGCTTC CACTGCAACA
 30361 AGTTGTTGCC GGCGGCTTTG TCCCAGCCAA TCAGCCTCGC CCCACTTCTC CCACCCCCAC
 30421 TGAAAATCAGC TACCTTAATC TAACAGGAGG AGATGACTGA CACCCCTAGAT CTAGAAAATGG
 30481 ACGGAATTAT TACAGAGCAG CGCCTGCTAG AAAGACGCAG GGCAGCGGCC GAGCAACAGC
 30541 GCATGAATCA AGAGCTCCAA GACATGGTTA ACTTGCACCA GTGCAAAGG GGTATCTTT
 30601 GTCTGGTAAA GCAGGCCAA GTCACCTACG ACAGTAATAC CACCGGACAC CGCCTTAGCT
 30661 ACAAGTTGCC ACCAACAGCGT CAGAAATTGG TGGTCATGGT GGGAGAAAAG CCCATTACCA
 30721 TAACTCAGCA CTCGGTAGAA ACCGAAGGCT GCATTCACTC ACCTTGTCAA GGACCTGAGG
 30781 ATCTCTGCAC CCTTATTAAG ACCCTGTGCG GTCTCAAAGA TCTTATTCCC TTTAACTAAT
 30841 AAAAAAAAT AATAAAGCAT CACTTACTTA AAATCAGTTA GCAAATTCT GTCCAGTTA
 30901 TTCAGCAGCA CCTCCCTTGCC CTCCTCCCAG CTCTGGTATT GCAGCTTCCT CCTGGCTGCA
 30961 AACTTTCTCC ACAATCTAAA TGGAATGTCA GTTTCCTCCCT GTTCCGTGTC ATCCGCACCC
 31021 ACTATCTTCA TGTGTTGCC GATGAAGCGC GCAAGACCGT CTGAAGATAC CTTCAACCCC
 31081 GTGTATCCAT ATGACACGGG AACCGGTCTC CCAACTGTGC CTTTTCTTAC TCCTCCCTT
 31141 GTATCCCCCA ATGGGTTTCA AGAGAGTCCC CCTGGGGTAC TCTCTTTGCG CCTATCGAA
 31201 CCTCTAGTTA CCTCCAATGG CATGCTTGCG CTCAAAATGG GCAACGGCCT CTCTCTGGAC
 31261 GAGGCCGGCA ACCCTTACCTC CCAAAATGTA ACCACTGTGA GCCCCACCTCT CAAAAAAACC
 31321 AAGTCAAACA TAAACCTGGG AATATCTGCA CCCCTCACAG TTACCTCAGA AGCCCTAACT
 31381 GTGGCTGCCG CGGCACCTCT AATGGTCGCG GGCAACACAC TCACCATGCA ATCACAGGCC
 31441 CCGCTAACCG TGCAAGACTC CAAACTTAGC ATTGGCACCC AAGGACCCCT CACAGTGTCA
 31501 GAAGGAAAGC TAGGCTTGCA AACATCAGGC CCCCTCACCA CAACCGATAG CAGTACCCCT
 31561 ACTATCACTG CCTCACCCCCC TCTAACTACT GCCACTGGT GCTTGGGCAT TGACTTGAAA
 31621 GAGCCCATT ATACACAAAA TGGAAAAGT GGACTAAAGT ACGGGGCTCC TTTGCATGTA
 31681 ACAGACGACC TAAACACTTT GACCGTAGCA ACTGGTCCAG GTGTACTAT TAATAAATACT
 31741 TCCCTGCAAA CTAAGTTAC TGGAGCCTTG GTTTTGATT CACAAGGCAA TATGCAACTT
 31801 AATGTAGCAG GAGGACTAAG GATTGATTCT CAAACAGAC GCCTTATACT TGATGTTAGT
 31861 TATCCGTTTG ATGCTCAAA CCAACTAAAT CTAAGACTAG GACAGGGCCC TCTTTTATA
 31921 AACTCAGCCC ACAACTTGGA TATTAACTAC AACAAAGGCC TTTACTTGT TACAGCTCA
 31981 ACAATTCCA AAAAGCTGAA GGTTAACCTA AGCACTGCCA AGGGGTTGAT GTTIGACGCT
 32041 ACAGCCATAG CCATTAATGC AGGAGATGGG CTTGAATTG GTTCACCTAA TGCACCAAAAC
 32101 ACAAAATCCCCC TCAAAACAAA AATTGGCCAT GGCCTAGAAT TTGATTCAA CAAGGCTATG
 32161 GTTCCCTAAAC TAGGAACTGG CCTTAGTTT GACAGCACAG GTGCCATTAC AGTAGGAAAC
 32221 AAAAATAATG ATAAGCTAAC TTTGTGGACC ACACCAAGCTC CATCTCCTAA CTGTAGACTA
 32281 AATGCAGAGA AAGATGCTAA ACTCACTTTG GTCTTAAACAA AATGTGGCAG TCAAATACTT
 32341 GCTACAGTTT CAGTTTTGGC TGTTAAAGGC AGTTTGGCTC CAATATCTGG AACAGTTCAA
 32401 AGTGCTCATC TTATTATAAG ATTTGACGAA AATGGAGTGC TACTAAACAA TTCCCTCTG
 32461 GACCCAGAAT ATTGGAACTT TAGAAATGGA GATCTTACTG AAGGCACAGC CTATACAAAC
 32521 GCTGTTGGAT TTATGCCAA CCTATCAGCT TATCCAAAAT CTCACGGTAA AACTGCCAA
 32581 AGTAACATTG TCAGTCAGT TTACTTAAAC GGAGACAAAA CTAAACCTGT AACACTAAC
 32641 ATTACACTAA ACGGTACACA GGAAACAGGA GACACAACCTC CAAGTGCATA CTCTATGTCA
 32701 TTTTCATGGG ACTGGTCTGG CCACAACTAC ATTATGAAA TATTTGCCAC ATCCTCTTAC
 32761 ACTTTTCAT ACATGCCAA AGAATAAAGA ATCGTTGTG TTATGTTCA ACGTGTCTTAT
 32821 TTTTCATTG CAGAAAATT TTACTTAAAC CAAGTCATT TTCAATTCACT AGTATAGCCC CACCACCA
 32881 TAGCTTATAC AGATCACCGT ACCTTAATCA AACTCACAGA ACCCTAGTAT TCAACCTGCC
 32941 ACCTCCCTCC CAACACACAG AGTACACAGT CCTTTCTCCC CGGCTGGCCT TAAAAGCAT
 33001 CATATCATGG GTAACAGACA TATTCTTAAAGG TGTTATATTC CACACGGTTT CCTGTGGAGC
 33061 CAAACGCTCA TCAGTGATAT TAATAAAACTC CCCGGCAGC TCACTTAAAGT TCATGTCGCT
 33121 GTCCAGCTGC TGAGCCACAG GCTGCTGTC AACTTACCGT TGCTTAAACGG GCGGCGAAGG
 33181 AGAAGTCCAC GCCTACATGG GGGTAGAGCT ATAATCGTGC ATCAGGATAG GCGGCTGGTG
 33241 CTGCAGCAGC GCGCGAATAA ACTGTCGCG CGCCTGCTCC GTCTCTGCAGG AATACAACAT
 33301 GGCAGTGGTC TCCTCAGCGA TGATTCGAC CGCCCGCAGC ATAAGGCGCC TTGTCCCTCCG
 33361 GGCACAGCAG CGCACCCCTGA TCTCACTTAA ATCAGCACAG TAACTGCAGC ACAGCACAC
 33421 AATATTGTTCA AAAATCCAC AGTGCAGG GCTGTATCCA AAGCTCATGG CGGGGACCCAC

FIGURE 21
(SHEET 10)

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33481 AGAACCCACG TGGCCATCAT ACCACAAGCG CAGGTAGATT AAGTGGCGAC CCCTCATAAA
 33541 CACGCTGGAC ATAAACATTA CCTCTTTGG CATGTTGTAA TTCACCACCT CCCGGTACCA
 33601 TATAAACCTC TGATTAACCA TGGGCCATC CACCACCATC CTAAACCAGC TGGCCAAAAC
 33661 CTGCCCGCCG GCTATACACT GCAGGGAACC GGGACTGGAA CAATGACAGT GGAGAGCCCA
 33721 GGACTCGTAA CCATGGATCA TCATGCTCGT CATGATATCA ATGTTGGCAC AACACAGGCA
 33781 CACGTGCATA CACTCCTCA GGATTACAAG CTCCCTCCGC GTTAGAACCA TATCCCAGGG
 33841 ACAAACCCAT TCCTGAATCA GCGTAAATCC CACACTGCAG GGAAGACCTC GCACGTAAC
 33901 CACGTTGTGC ATTGTCAAAG TGTTACATTC GGGCAGCAGC GGATGATCCT CCAGTATGGT
 33961 AGCGCGGGTT TCTGTCTCAA AAGGAGGTAG ACGATCCCTA CTGTAACGGAG TGCGCCGAGA
 34021 CAACCGAGAT CGTGTGGTC GTAGTGTCA GCAAATGGA ACAGCCGGACG TAGTCATATT
 34081 TCCTGAAGCA AAACCCAGGTG CGGGCGTGC AAACAGATCT GCGTCTCCGG TCTCGCCGCT
 34141 TAGATCGTC TGTGTAGTAG TTGTAGTATA TCCACTCTCT CAAAGCATCC AGGCGCCCCC
 34201 TGGCTTCGGG TTCTATGTAA ACTCCTTCAT GCGCCGCTGC CCTGATAACCA TCCACCAACCG
 34261 CAGAATAAGC CACACCCAGC CAACCTACAC ATTCTGTTCTG CGAGTCACAC ACGGGAGGAG
 34321 CGGGAAGAGC TGGAGAAACC ATGTTTTTTT TTATTCCTA AAAGATTATC CAAAACCTCA
 34381 AAATGAAGAT CTATTAAGTG AACCGCCTCC CCTCCGGTGG CGTGGTCAAA CTCTACAGCC
 34441 AAAGAACAGA TAATGGCATT TGTAAGATGT TGCAACATGG CTTCACAAAG GCAAACGGCC
 34501 CTCACGTCGA AGTGGACGTA AAGGCTAAAC CCTTCAGGGT GAATCTCCTC TATAAACATT
 34561 CCAGCACCTT CAACCATGCC CAAATAATTTC TCATCTGCC ACCTTCTCAA TATATCTCTA
 34621 AGCAAATCCC GAATATTAAG TCCGGCCATT GTAAAAATCT GCTCCAGAGC GCCCTCCACC
 34681 TTCAGCCTCA AGCAGCGAAT CATGATTGCA AAAATTCAAGG TTCTCACAG ACCTGTATAA
 34741 GATTCAAAAG CGGAACATTA ACAAAAATAC CGCGATCCCG TAGGTCCTT CGCAGGGCCA
 34801 GCTGAACATA ATCGTGCAGG TCTGCACGGA CCAGCGCGC CACTTCCCCG CCAGGAACCT
 34861 TGACAAAAGA ACCCACACTG ATTATGACAC GCATACTCGG AGCTATGCTA ACCAGCGTAG
 34921 CCCCCGATGTA AGCTTGTG TGATGGCGC GATATAAAAT GCAAGGTGCT GCTCAAAAAA
 34981 TCAGGCAAAG CCTCGCGCAA AAAAGAAAGC ACATCGTAGT CATGCTCATG CAGATAAAGG
 35041 CAGGTAAAGCT CGGAACCCAC CACAGAAAAA GACACCAATT TTCTCTCAA CATGTCTGCG
 35101 GGTTCCTGCA TAAACACAAA ATAAAATAAC AAAAAAAACAT TTAAACATTA GAAGCCTGTC
 35161 TTACAACAGG AAAAACAAACC CTTATAAGCA TAAGACGGAC TACGGCCATG CCGGCGTGAC
 35221 CGTAAAAAAA CTGGTCACCG TGATTTAAAAA GCACCAACCGA CAGCTCCTCG GTCATGTCCG
 35281 GAGTCATAAT GTAAGACTCG GTAAACACAT CAGGTTGATT CATCGGTCAG TGCTAAAAAG
 35341 CGACCGAAAT AGCCCCGGGG AATACATACC CGCAGCGTA GAGACAACAT TACAGCCCCC
 35401 ATAGGAGGTA TAACAAAATT AATAGGAGAG AAAAACACAT AAACACCTGA AAAACCTCC
 35461 TGCCTAGGCA AAATAGCACC CTCCCGCTCC AGAACACAT ACAGCGCTTC ACAGCGCAG
 35521 CCTAACAGTC AGCCTTACCA GTAAAAAAGA AAACCTATTA AAAAAACACC ACTCGACACG
 35581 GCACCAGCTC AATCAGTCAC AGTGTAAAAA AGGGCCAAGT GCAGAGCGAG TATATATAGG
 35641 ACTAAAAAAT GACGTAACGG TTAAAGTCCA CAAAAACAC CCAGAAAAC GCACGCGAAC
 35701 CTACGCCAG AAACGAAAGC CAAAAAACCC ACAACTTCCT CAAATCGTCA TTTCGTTTT
 35761 CCCACGTTAC GTAACTTCCT ATTITAAGAA AACTACAATT CCCAACACAT ACAAGTTACT
 35821 CCGCCCTAAA ACCTACGTCA CCCGCCCCGT TCCCAAGGCC CGCGCCACGT CACAAACTCC
 35881 ACCCCCTCAT TATCATATTG GCTTCATTC CAAATAAGGT ATATTATTGA TGATG

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FIGURE 21
(SHEET 11)

ad5

33166

LOCUS KD1 **33592 bp** **DNA** **SYN** **28-APR-1999**
DEFINITION KD1
ACCESSION KD1
KEYWORDS
SOURCE Unknown.
ORGANISM Unknown
Unclassified.
REFERENCE 1 (bases 1 to 33592)
AUTHORS Self
JOURNAL Unpublished.
FEATURES Location/Qualifiers
CDS 1..33592
/gene="KD1"
/product="KD1"
BASE COUNT 7744 a 9470 c 9285 g 7093 t
ORIGIN

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1 CATCATCAAT AATATAACCTT ATTTTGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
61 TTGTGACGTG GCGCGGGGCG TGGGAACGGG GCGGGTGACG TAGTAGTGTG GCGGAAGTGT
121 GATGTTGCAA GTGTGGCGGA ACACATGTAA GCGACGGATG TGGCAAAAGT GACGTTTTTG
181 GTGTGCGCCG GTGTACACAG GAAGTGCACAA TTTCTCGC GGGAAAACGT AATAAGAGGA
241 TAAATTGGG CGTAACCGAG TAAGATTGG CCATTTTCGC GGGAAAACGT AATAAGAGGA
301 AGTGAATCT GAATAATTTT GTGTTACTCA TAGCGCGTAA TATTTGTCTA GGGCCGCGGG
361 GACTTGTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTTT CTCAGGTGTT TTCCCGGTC
421 CGGGTCAAAG TTGGCGTTT ATTATTATAG TCAGCTGACG TGTAGTGTAT TTATACCCGG
481 TGAGITCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTCTCC TCCGAGCCGC
541 TCCGACACCG GGACTGAAA TGAGACATGA GGTACTGGCT GATAATCTTC CACCTCCTAG
601 CCATTGAA CCACCTACCC TTCACGAACG GTATGATTTA GACGTGACGG CCCCCGAAGA
661 TCCCAACGAG GAGGCGGTTT CGCAGATTT TCCCGACTCT GTAATGTGG CGGTGCAGGA
721 AGGGATTGAC TTACTCACTT TTCCGCCGC GCCCGGTCTC CCGGAGCCGC CTCACCTTTC
781 CCGGAGCCC GAGCAGCCGG AGCAGAGAGC CTGGGTCCG GTTGCCACG AGGCTGGCTT
841 TCCACCCAGT GACGACGAGG ATGAAGAGGG TGAGGAGTT GTGTTAGATT ATGTGGAGCA
901 CCCGGGCAC GGTTGCAGGT CTGTCATTA TAACCGGGAG AATACGGGG ACCCAGATAT
961 TATGTGTG TGTTGCTATA TGAGGACCTG TGCGATGTT GTCTACAGTA AGTAAAATT
1021 ATGGCAGTG GGTGATAGAG TGGTGGTTT GGTGTTGAA TTTTTTTTTT AATTTTTACA
1081 GTTTGTGGT TTAAAGAATT TTGTATTGTG ATTTTTTAA AAGGTCTGT GTCTGAACCT
1141 GAGCTGAGC CCGAGCCAGA ACCGGAGCT GCAAGACCTA CCCGCGGTCC TAAAATGGCG
1201 CCTGCTATCC TGAGACGCC GACATCACCT GTGTCTAGAG AATGCAATAG TAGTACGGAT
1261 AGCTGTGACT CCGGTCTTC TAACACACCT CCTGAGATAAC ACCCGGTGGT CCCGCTGTGC
1321 CCCATTAAAC CAGTTGCCGT GAGAGTTGGT GGGCGTCGCC AGGCTGTGGA ATGTATCGAG
1381 GACTTGCTTA ACGAGCCTGG GCAACCTTG GACTTGAGCT GTAAACGCC CAGGCCATAA
1441 GGTGTAAACC TGTGATTGGC TGTGTTGTTA ACGCCCTTGT TTGCTGAATG AGTGTGATGTA
1501 AGTTAATAA AGGGTGAGAT AATGTTAAC TTGCGATGGCG TGTTAAATGG GGGGGGGCTT
1561 AAAGGGTATA TAATGCCCG TGGGCTAATC TTGGTTACAT CTGACCTCAT GGAGGCTTGG
1621 GAGTTTTGG AAGATTTTC TGCTGTGCGT AACTTGCTGG AACAGAGCTC TAACAGTACC
1681 TCTTGGTTT GGAGGTTCT GTGGGGCTCA TCCCAGGCAA AGTTAGTCTG CAGAATTAAG
1741 GAGGATTACA AGTGGGAATT TGAAGAGCTT TTGAAATCTC GTGGTGTAGCT GTTGTGATTCT
1801 TTGAATCTGG GTCACCAAGGC GCTTTCTCAA GAGAAGGTCA TCAAGACTTT GGATTTTCC
1861 ACACCGGGGC GCGCTGCCGC TGCTGTGCT TTTTGAGTT TTATAAAGGA TAAATGGAGC
1921 GAAGAAACCC ATCTGAGCGG GGGGTACCTG CTGGATTTTC TGCCATGCA TCTGTGGAGA
1981 GCGGTTGTGA GACACAAGAA TCGCCCTGCTA CTGTTGCTT CCGTCCGCC GGGATAATA
2041 CCGACGGAGG AGCAGCAGCA GCAGCAGGAG GAAGCCAGGC GGCGGCGGCA GGAGCAGAGC
2101 CCATGGAACC CGAGAGCCGG CCTGGACCTC CGGGAATGAA TGTTGTACAG GTGGCTGAAC
2161 TGTATCCAGA ACTGAGACGC ATTTTGACAA TTACAGAGGA TGGGCAAGGGG CTAAAGGGGG
2221 TAAAGAGGGG GCGGGGGGCT TGTGAGGCTA CAGAGGAGGC TAGGAATCTA GCTTTTAGCT
2281 TAATGACCAAG ACACCGTCT GAGTGTATTA CTTTCAACA GATCAAGGGAT AATTGCGCTA
2341 ATGAGCTTGA TCTGCTGGCG CAGAAGTATT CCATAGAGCA GCTGACCACT TACTGGCTGC
2401 AGCCAGGGGA TGATTTGAG GAGGCTATTA GGGTATATGC AAAGGTGGCA CTTAGGCCAG

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kd1

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FIGURE 22
(SHEET 1)

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2461 ATTGCAAGTA CAAGATCAGC AAACTTGTAA ATATCAGGAA TTGTTGCTAC ATTTCTGGGA
 2521 ACGGGGCCGA GGTGGAGATA GATA CGGAGG ATAGGGTGGC CTTTAGATGT AGCATGATAA
 2581 ATATGTGCC GGGGGTGCCTT GGCATGGACG GGGTGGTTAT TATGAATGTA AGGTTTACTG
 2641 GCCCCAATT TAGCGGTACG GTTTTCTGG CCAATACCAA CCTTATCCTA CACGGTGAA
 2701 GCTTCTATGG GTTTAACAAAT ACCTGTGTGG AAGCCTGGAC CGATGTAAGG GTTCGGGCT
 2761 GTGCCTTTA CTGCTGCTGG AAGGGGGTGG TGTGTCGCC CAAAAGCAGG GCTTCATTAA
 2821 AGAAATGCCT CTTTGAAAGG TGTACCTTGG GTATCCTGTC TGAGGGTAAC TCCAGGGTGC
 2881 GCCACAATGT GGCCTCCGAC TGTGGTTGCT TCATGCTAGT GAAAAGCGTG GCTGTGATTA
 2941 AGCATAAACAT GGTATGTGGC AACTGCGAGG ACAGGGCCTC TCAGATGCTG ACCTGCTCGG
 3001 ACGGCAACTG TCACCTGCTG AAGACCATTG ACGTAGCCAG CCACCTCTCGC AAGGCCTGGC
 3061 CAGTGTGTTGA GCATAACATA CTGACCCGCT GTTCCTTGCA TTTGGGTAAC AGGAGGGGG
 3121 TGTTCCTTAC TTACCAATGC AATTGAGTC ACACTAAGAT ATTGCTTGAG CCCGAGAGCA
 3181 TGTCCAAGGT GAACCTGAAC GGGGTGTTG ACATGACCAT GAAGATCTGG AAGGTGCTGA
 3241 GGTACGATGA GACCCGCAAC AGGTGCGAGAC CCTGCGAGTG TGGCGGTAAA CATATTAGGA
 3301 ACCAGCCTGT GATGCTGGAT GTGACCCGAGG AGCTGAGGCC CGATCACTTG GTGCTGGCCT
 3361 GCACCCGCGC TGAGTTTGGC TCTAGCGATG AAGATACAGA TTGAGGTACT GAAATGTGTG
 3421 GGCGTGGCCTT AAGGGTGGGA AAGAATATAT AAGGGTGGGG TCTTATGTAG TTTTGTATCT
 3481 GTTTTGACGC AGCCGCCGCC GCCATGAGCA CCAACTCGTT TGATGGAAGC ATTGTGAGCT
 3541 CATATTTGAC AACCGCGATG CCCCCATGGG CCGGGGTGCG TCAGAATGTC ATGGGCTCCA
 3601 GCATTGATGG TCGCCCCGTC CTGCCCCGCAA ACTCTACTAC CTGACCTAC GAGACCGTGT
 3661 CTGGAACCGC GTTGGAGACT GCAGCCTCCG CCGCCCTTC AGCCCTTGCA GCCACCCGCC
 3721 GCGGGATTGT GACTGACTTT GTTTCTGCA GCGCCCTTGCA AAGCAGTGCA GCTTCCCGTT
 3781 CATCCGCCCG CGATGACAAG TTGACGGCTC TTTGGCACA ATTGGATTCT TTGACCCGGG
 3841 AACTTAATGT CGTTTCTCAG CAGCTGTTGG ATCTGCGCCA GCAGGTTTCT GCCCTGAAGG
 3901 CTTCCTCCCC TCCCAATGCG GTTTAAACAA TAAATAAAAA ACAGACTCT GTTGGATT
 3961 GGATCAAGCA AGTGTCTTGC TGTCTTTATT TAGGGGTTTT GCGCGCGCG TAGGCCCGGG
 4021 ACCAGCGGTC TCGGTGTTGG AGGGTCCTGT GTATTTC CAGGACGTGG TAAAGGTGAC
 4081 TCTGGATGTT CAGATACATG GGCAATAAGG CGTCTCTGGG GTGGAGGTAG CACCACTGCA
 4141 GAGCTTCATG CTGCGGGGTG GTGTTGTAGA TGATCCAGTC GTAGCAGGAG CGCTGGCGT
 4201 GGTGCCCTAAA AATGTCTTTC AGTAGCAAGC TGATGCCAG GGGCAGGCC TTGGTGTAG
 4261 TGTTCACAAA GCGGTTAACG TGGGATGGGT GCATACGTGG GGATATGAGA TGCACTTGG
 4321 ACTGTATTT TAGGTTGGCT ATGTTCCAG CCATATCCCT CGGGGGATTC ATGTTGTCA
 4381 GAACCAACAG CACAGTGTAT CCGGTGCACT TGGGAAATT GTCATGTAGC TTAGAAGGAA
 4441 ATGCGTGAA GAACCTGGAG ACGCCCTTGT GACCTCAAG ATTTCCATG CATTGCTCCA
 4501 TAATGATGGC AATGGGCCCA CGGGCGGCCG CCTGGCGAA GATATTTCTG GGATCACTAA
 4561 CGTCATAGTT GTGTTCCAGG ATGAGATCGT CATAGGCCAT TTTTACAAAG CGGGGGCGGA
 4621 GGGTGCCAGA CTGCGGTATA ATGGTTCCAT CGGGCCCAGG GCGTAGTTA CCCTCACAGA
 4681 TTTGCATTTC CCACGGCTTG AGTTCAGATG GGGGGATCAT GTCTACCTGC GGGCGATGA
 4741 AGAAAACGGT TTCCGGGTTA GGGGAGATCA GCTGGGAAGA AAGCAGGTTG CTGAGCAGCT
 4801 GCGACTTAC GCAGCCGGTG GGGCCGTAAA TCACACCTAT TACCGGGTGC AACTGGTAGT
 4861 TAAGAGAGCT GCAGCTGCCG TCATCCCTGA GCAGGGGGC CACTTCGTTA AGCATGTCCC
 4921 TGACTCGCAT GTTTCCCTG ACCAAATCCG CCAGAAGGCG CTCGCCGCC AGCGATAGCA
 4981 GTTCTTGCAA GGAAGCAAAG TTTTCAACG GTTGAGACC GTCCGCCGTA GGCATGCTTT
 5041 TGAGCGTTTG ACCAAGCAGT TCCAGGCGGT CCCACAGCTC GGTCACCTGC TCTACGGCAT
 5101 CTCGATCCAG CATATCTCCT CGTTTCGCGG GTTGGGGCGG CTTTCGCTGT ACGGCAGTAG
 5161 TCGGTGCTCG TCCAGACGGG CCAGGGTCAT GTCTTCCAC GGGCGCAGGG TCCTCGTCAG
 5221 CGTAGCTCTGG GTCACGGTGA AGGGGTGCGC TCCGGGCTGC GCGCTGGCCA GGGTGGCGCTT
 5281 GAGGCTGGTC CTGCTGGTGC TGAAGCGCTG CCGGCTCTCG CCCTGCGCGT CGGCCAGGTA
 5341 GCATTGACCT ATGGTGTCAAT AGTCCAGCCC CTCCGCGCGC TGGCCCTTG CGCGCAGCTT
 5401 GCCCCGGAG GAGGGCGCCGC ACGAGGGGCA GTGCAGACTT TTGAGGGCGT AGAGCTTGGG
 5461 CGCGAGAAAT ACCGATTCCG GGGAGTAGGC ATCCGCGCCG CAGGCCCGC AGACGGTCTC
 5521 GCATTCCACG AGCCAGGTGA GCTCTGGCCG TTCGGGGTCA AAAACCAGGT TTCCCCCATG
 5581 CTTTTGATG CGTTTCTTAC CTCTGGTTTC CATGAGCCGG TGTCCACGCT CGGTGACGAA
 5641 AAGGCTGTCC GTGTTCCCGT ATACAGACCTT GAGAGGCCCTG TCCTCGAGCG GTGTTCCGG
 5701 GTCCCTCTCG TATAGAAACT CGGACCACTC TGAGACAAAG GCTCGCGTCC AGGCCAGCAC
 5761 GAAGGAGGCT AAGTGGGAGG GGTAGCGGTG TTGTCCTACT AGGGGGTCCA CTCGCTCCAG
 5821 GGTGTGAAGA CACATGTCGC CCTCTTCGGC ATCAAGGAAG GTGATTGGTT TGTAGGTGTA

FIGURE 22
(SHEET 2)

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5881 GGCCACGTGA CCGGGTGTTC CTGAAGGGGG GCTATAAAAG GGGGTGGGGG CGCGTTCGTC
 5941 CTCACTCTCT TCCGCATCGC TGTCTGCGAG GGCCAGCTGT TGGGGTGAGT ACTCCCTCTG
 6001 AAAAGCGGGC ATGACTTCTG CGCTAAGATT GTCAGTTTCC AAAAACGAGG AGGATTGAT
 6061 ATTACACCTGG CCCGGGGTGA TGCTTTGAG GGTGGCCGCA TCCATCTGGT CAGAAAAGAC
 6121 AATCTTTTG TTGTCAGACT TGGTGGCAA CGACCCGTAG AGGGCGTTGG ACAGCAACTT
 6181 GGCGATGGAG CGCAGGGTTT GTTTTGTGCG GCGATCGGCG CGCTCCTTGG CCGCGATGTT
 6241 TAGCTGCACG TATTCGCGCG CAACGCACCG CCATTCGGGA AAGACGGTGG TGCGCTCGTC
 6301 GGGCACCAAGG TGACACGCC ACCCGCGGT GTGCAGGGTG ACAAGGTCAA CGCTGGTGGC
 6361 TACCTCTCCG CGTAGGCCT CGTTGGTCCA GCAGAGGCGG CGGCCCTTGC GCGAGCAGAA
 6421 TGGCGTAGG GGGCTAGCT GCGTCTCGTC CGGGGGTCT CGGTCCACGG TAAAGACCCC
 6481 GGGCAGCAGG CGCGCGTCGA AGTAGTCTAT CTGCACTCT TGCAAGTCTA GCGCCTGCTG
 6541 CCATGCGCGG GCGGCAAGCG CGCGCTCGTA TGGGTGAGT GGGGGACCCC ATGGCATGGG
 6601 GTGGGTGAGC GCGGAGGCCT ACATGCCGCA ATGTCGTAAC AGCTAGAGGG GCTCTCTGAG
 6661 TATTCCAAGA TATGAGGGT AGCATCTTC ACCCGGGATG CTGGCGCGA CGTAATCGTA
 6721 TAGTTCTGTC GAGGGAGCGA GGAGGTGGGG ACCGAGGTTG CTACGGGCGG GCTGCTCTGC
 6781 TCAGAACACT ATCTGCCTGA AGATGGCATG TGAGTTGGAT GATATGGTTG GACGCTGGAA
 6841 GACGTTGAAG CTGGCGTCTG TGAGACCTAC CGCGTCACCG ACCAAGGAGG CGTAGGAGTC
 6901 GCGCAGCTTG TTGACCAGCT CGGCGGTGAC CTGCACTCT GAGGGCGCAGT AGTCCAGGGT
 6961 TTCCCTGATG ATGTCATACT TATCTCTGTC CTTTTTTTTC CACAGCTCGC GGTTGAGGAC
 7021 AAACCTCTCG CGGCTTTCC AGTACTCTG GATCGGAAAC CGCTGGCCT CCGAACGGTA
 7081 AGAGCCTAACG ATGTAAGACT GTTGAACGGG CTGGTAGGCG CAGCATTCCCT TTTCTACGGG
 7141 TAGCGCTAT GCCTGCGCGG CCTTCCGGAG CGAGGTGTGG GTGAGCGCAA AGGTGTCCCT
 7201 GACCATGACT TTGAGGTTACT GGTATTGAA GTCAGTGTG TCGCATCCGC CCTGCTCCCA
 7261 GAGAAAAAG TCCGTGCGCT TTTGGAACG CGGATTGCG AGGGCGAAGG TGACATCGTT
 7321 GAAGAGTATC TTTCCCGCGC GAGGCATAAA GTTGCCTGTG ATGCGGAAGG GTCCCCGGCAC
 7381 CTCGGAACCG TTGTTAATTAA CCTGGCGGGC GAGCACGATC TCGTCAAAGC CGTTGATGTT
 7441 GTGGCCACA ATGTAAGTT CCAAGAAGCG CGGGATGCCC TTGATGGAAG GCAATTGTTT
 7501 AAGTTCTCG TAGGTGAGCT CTTCAGGGGA GCTGAGCCCG TGCTCTGAAA GGGCCAGTC
 7561 TGCAAGATGA GGGTTGGAAG CGACGAATGA GCTCCACAGG TCACGGGCCA TTAGCATTG
 7621 CAGGTGGTCG CGAAAGGTCC TAAACTGGCG ACCTATGGCC ATTTTTCTG GGGTGATGCA
 7681 GTAGAAGGTA AGCGGGTCTT GTTCCCAGCG GTCCCACCCA AGGTTCGCGG CTAGGTCTCG
 7741 CGCGCAGTC ACTAGAGGCT CATCTCCGCC GAACTTCATG ACCAGCATGA AGGGCACGAG
 7801 CTGCTTCCCA AAGGCCCCCA TCCAAGTATA GGTCTCTACA TCGTAGGTGA CAAAGAGACG
 7861 CTCGGTGCAGA GGATGCGAGC CGATCGGGAA GAACTGGATC TCCCGCCACC AATTGGAGGA
 7921 GTGGCTATTG ATGTGGTGAA AGTAGAAAGTC CCTGCGACGG GCCGAACACT CGTGTGGCT
 7981 TTTGAAAAA CGTGCAGT ACTGGCAGCG GTGACCGGGC TGACATCCT GCACGAGGTT
 8041 GACCTGACGA CCGCGCACAA GGAAGCAGAG TGGGAATTG AGCCCCCTCGC CTGGCGGGTT
 8101 TGGCTGGTG TCTTCTACTT CGGCTGCTTG TCCCTGACCG TCTGGCTGCT CGAGGGGAGT
 8161 TACGGTGGAT CGGACCACCA CGCCGCGCGA GCCCCAAAGTC CAGATGTCCG CGCGCGCGG
 8221 TCGGAGCTTG ATGACAACAT CGCGCAGATG GGAGCTGTCC ATGGTCTGGA GCTCCCCGG
 8281 CGTCAGGTCA GGGGGAGCT CCTGCAGGTT TACCTCGCAT AGACGGGTCA GGGCGGGG
 8341 TAGATCCAGG TGATACCTAA TTCCAGGGG CTGGTGGTG GCGCGTGCAGA TGGCTTGCAA
 8401 GAGGCCGCAT CCCCCGGCG CGACTACGGT ACCCGCGGGC GGGCGGTGGG CGCGGGGGGT
 8461 GTCCCTGGAT GATGCATCTA AAAGCGGTGA CGCGGGGAGG CCCCCGGAGG TAGGGGGGC
 8521 TCCGGACCCG CGGGGAGAGG GGGCAGGGGA ACGTCGGCGC CGCGCGGGGG CAGGAGCTGG
 8581 TGCTGCGCG TGAGGTGGCT GCGGAACGGC ACGACCGGGC GTTGAATCTC CTGAATCTGG
 8641 CGCCTCTGCG TGAAGACGAC GGGCCCGGTG AGCTTGAGCC TAAAAGAGAG TTCGACAGAA
 8701 TCAATTCTGG TGTCTGGTGC CGCGGGCTGG CGCAAAATCT CCTGCACTG TCCCTGAGTTG
 8761 TCTTGATAGG CGATCTCGGC CATGAACATG TCGATCTCTT CCTCCCTGGAG ATCTCCGCT
 8821 CGGGCTCGCT CCACGGTGGC CGGGAGGTG TTGGAAATGC GGGCCATGAG CTGCGAGAAG
 8881 GCGTTGAGGC CTCCCTCGTT CGAGACGCGG CTGTAGACCA CGCCCCCTTC GGCATCGCGG
 8941 GCGCGCATGA CCACCTGCGC GAGATTGAGC TCCACGTGC GGGCGAAGAC GCGTAGTTT
 9001 CGCAGGCCT GAAAGAGGTA GTTGGGGTG GTGGGGTGT GTTCTGCCAC GAAGAAGTAC
 9061 ATAACCCAGC GTCGCAACGT GGATTGTTG ATATCCCCA AGGCCTCAAG GCGCTCCATG
 9121 GCCTCGTAGA AGTCCACGGC GAAAGTTGAAA AACTGGGAGT TGCGCGCCGA CACGGTTAAC
 9181 TCCCTCTCCA GAAGACGGAT GAGCTCGGGC ACAGTGTGCG GCACCTCGCG CTCAAAGGCT
 9241 ACAGGGGCCT TTTCTCTTC TTCAATCTCC TCTTCCATAA GGGCCTCCCC TTCTTCTTCT

FIGURE 22
(SHEET 3)

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9301 TCTGGCGGCG GTGGGGGAGG GGGGACACGG CGGCGACGAC GGCGCACCGG GAGGCAGTCG
 9361 ACAAAGCGCT CGATCATCTC CCCGCGGCGA CGGCGCATGG TCTCGGTGAC GGCGCGGGCG
 9421 TTCTCGCGGG GGCGCAGTTG GAAGACGCCG CCCGTATGT CCCGGTTATG GTTGGCGGG
 9481 GGGCTGCCAT CGGGCAGGGGA TACGGCGCTA ACGATGCATC TCAACAATTG TTGTGTAGGT
 9541 ACTCCGCCCG CGAGGGACCT GAGCGAGTCC GCATCGACCG GATCGAAAAA CCTCTCGAGA
 9601 AAGGCCTCTA ACCAGTCACA GTCGCAAGGT AGGCTGAGCA CCGTGGCGGG CGGCAGCGGG
 9661 CGGGTCTCGG GTTGTCTCT GCGGGAGGTG CTGCTGATGA TGTAATTAAA TAGGCAGTC
 9721 TTGAGACGGC GGATGGTCGA CAGAAGCACC ATGTCCTTGG GTCCGGCTG CTGAATGCGC
 9781 AGGCGGTCGG CCATGCCCA GGCTTCGTT TGACATGGC GCAGGTCTT GTAGTAGTCT
 9841 TGATGAGCC TTTCTACCGG CACTTCTTCT TCTCCTCT CTTGTCCTGC ATCTCTGCA
 9901 TCTATCGCTG CGGGCGGGC GGAGTTTGGC CGTAGGTGGC GCCCTCTTCC TCCCCTGCGT
 9961 GTGACCCCCGA AGCCCCCTCAT CGGCTGAAGC AGGGCTAGGT CGGCGACAAAC GCGCTCGGCT
 10021 AATATGGCCT GCTGCACCTG CGTGAGGGTA GACTGGAAGT CATCCATGTC CACAAAGCGG
 10081 TGGTATGCGC CCGTGTGAT GGTGTAAGTG CAGTTGGCA TAACGGACCA GTTAACGGTC
 10141 TGGTGAACCG GCTGCGAGAG CTCGGTGTAC CTGAGACCGC AGTAAGCCCT CGAGTCAAAT
 10201 ACGTAGTCGT TGCAAGTCCG CACCAAGGTAC TGTTATCCA CCAAAAGTG CGGCGGGCGC
 10261 TGGCGGTAGA GGGGCCAGCG TAGGGTGGCC GGGGCTCCCG GGGCGAGATC TTCCAACATA
 10321 AGGCGATGAT ATCCGTAGAT GTACCTGGAC ATCCAGGTGA TGCCGGCGGC GTGTTGGAG
 10381 GCGCGCGGA AGTCGCGGAC GCGGTTCCAG ATGTTGCGCA GCGGCAAAAA GTGCTCCATG
 10441 GTCGGGACGC TCTGGCCGGT CAGGCGCGCG CAATCGTTGA CGCTCTAGCG TGCAAAAGGA
 10501 GAGCCTGTA GCGGGCACTC TTCCGTGGTC TGGTGGATAA ATTCGCAAGG GTATCATGGC
 10561 GGACGACCCG GGTCGAGGC CCGTATCCGG CCGTCCCGG TGATCCATGC GTTACCGCC
 10621 CGCGTGTGCGA ACCCAGGTGT GCGACGTCAG ACAACGGGGG AGTGCTCCTT TTGGCTTCC
 10681 TCCAGGCCCG GCGGCTGCTG CGCTAGTTT TTGGGCAACT GGGCGCGCG AGCGTAAGCG
 10741 GTTACGGCTGG AAAGCGAAAG CATTAAAGTGG CTCGCTCCCT GTAGGGGGAG GTTATTTC
 10801 CAAGGGTTGA GTCGGGGAC CCCCCGGTTCG AGTCTCGGAC CGGCGGACT GCGGCGAACG
 10861 GGGGTTTGC CCCCCGTCA GCAAGACCCCC GCTTGAAT TCTCCGGAA ACAGGGACGA
 10921 GCCCCCTTTT TGCTTTCCC AGATGCAATC GGTGCTGCGG CAGATGCGCC CCCCTCTCA
 10981 GCAGCGGCAA GAGCAAGAGC AGCGGGAGAC ATGCAGGGCA CCTCTCCCTC CTCCCTACCG
 11041 GTCAAGGAGGG GCGACATCCG CGGTTGACGC GGCAGCAGAT GGTGATTACG AACCCCCCG
 11101 GCGCCGGGCC CGGCACCTACC TGGACTTGGG GGAGGGCGAG GGCCTGGCGC GGCTAGGAGC
 11161 GCCCCCTCTCT GAGCGGTACC CAAGGGTGC GCTGAAGCGT GATACGCGTG AGGCGTACGT
 11221 GCGCGGGCAG AACCTGTTTC GCGACCGCGA GGGAGAGGAG CCCGAGGAGA TGCGGGATCG
 11281 AAAGTTCCAC GCAGGGCGCG AGCTGCGCA TGGCTGAAT CGCGAGCGGT TGCTGCGCGA
 11341 GGAGGACTTT GAGCCCACG CGCGAACCGG ATTAGTCCC CGCGCGCGAC ACGTGGCGGC
 11401 CGCCGACCTG GTAACCGCAT ACGAGCAGAC GGTGAACCG GAGATTAACT TTCAAAAAG
 11461 CTTAACACAC CACGTGCGTA CGCTTGTGGC GCGCGAGGAG GTGGCTATAG GACTGATGCA
 11521 TCTGTGGGAC TTTGTAAGCG CGCTGGAGCA AAACCCAAT AGCAAGCCGC TCATGGCGCA
 11581 GCTGTTCCCTT ATAGTGCAGC ACAGCAGGGG CAACGAGGCA TTCAGGGATG CGCTGCTAAA
 11641 CATAGTAGAG CCCGAGGGCC GCTGGCTGCT CGATTTGATA AACATCCTGC AGAGCATAGT
 11701 GGTGCAGGAG CGCAGCTTGA GCCTGGCTGA CAAGGTGGCC GCCATCAACT ATTCCATGCT
 11761 TAGCCTGGGC AAGTTTACG CCGCAAGAT ATACCATAAC CCTTACGTT CCATAGACAA
 11821 GGAGGTAAG ATCGAGGGGT TCTACATGCG CATGGCGCTG AAGGTGCTTA CTTTGAGCGA
 11881 CGACCTGGGC GTTATCGCA ACGAGCGCAT CCACAAGGC GTGAGCGTGA GCCGGCGGG
 11941 CGAGCTCAGC GACCGCGAGC TGATGCACAG CCTGCAAAGG GCCCTGGCTG GCACGGCAG
 12001 CGCGATAGA GAGGGCGAGT CCTACTTTGA CGCGGGCGCT GACCTGCGCT GGGCCCAAG
 12061 CCGACCGGCC CTGGAGGCAG CTGGGGCCGG ACCTGGCTG GCGGTGGCAG CGCGCGCGC
 12121 TGGCAACGTC GGCAGCGTGG AGGAATATGA CGAGGACGAT GAGTACGAGC CAGAGGACGG
 12181 CGAGTACTAA GCGGTGATGT TTCTGATCAG ATGATGCAAG ACACGCAACGGA CCCGGCGGTG
 12241 CGGGCGGCGC TGCAGAGCCA GCGTCCGGC CTTAACCTCA CGGACGACTG GCGCCAGGTC
 12301 ATGGACCGCA TCATGTCGCT GACTGCGCG AATCCTGACG CGTTCCGGCA CGAGCCGCAG
 12361 GCCAACCGGC TCTCCGCAAT TCTGGAAGCG GTGGTCCCGG CGCGCGCAA CCCCACGCAC
 12421 GAGAAGGTGC TGGCGATCGT AAACCGCGCTG GCGAAAACA GGGCCATCCG GCGGACCGAG
 12481 CGCCGGCTGG TCTACGACGC GCTGCTTCAG CGCGTGGCTC GTTACAAACAG CGGCAACGTG
 12541 CAGACCAACC TGGACCGGCT GTGGGGGGAT GTGCGCGAGG CGTGGCGCGA GCGTGAGCGC
 12601 CGCGAGCGAC AGGGCAACCT GGGCTCCATG GTTGCACTAA ACACGCTTCCCT GAGTACACAG
 12661 CCCGCCAACG TGCGCGGGGG ACAGGAGGAC TACACCAACT TTGTGAGCGC ACTGCGGCTA

12721 ATGGTGAATG AGACACCGCA AAGTGAGGTG TACCAAGTCTG GGCCAGACTA TTTTTCCAG
 12781 ACCAGTAGAC AAGGCCTGCA GACCGTAAAC CTGAGCCAGG CTTTCAAAAA CTTGCAGGGG
 12841 CTGTGGGGGG TGCGGGCTCC CACAGGCAC CGCGCGACCG TGCTCTAGCTT GCTGACGCC
 12901 AACTCGGCCC TGTGCTGCT GCTAATAGCG CCCTTCACGG ACAGTGGCAG CGTGTCCCG
 12961 GACACATACC TAGGTCACCT GCTGACACTG TACCGCGAGG CCATAGGTCA GGCGCATGTG
 13021 GACGAGCATA CTTTCAGGA GATTACAAGT GTCAAGCCGCG CGCTGGGGCA GGAGGACACG
 13081 GGCAGCTGG AGGCAACCC AAACCTACCTG CTGACCAACC GGCGGCAGAA GATCCCCTCG
 13141 TTGACACAGTT TAAACAGCGA GGAGGAGCGC ATTTTGCCT ACGTGAGCAG GAGCGTGAGC
 13201 CTTAACCTGA TGCGCGACGG GGTAAACGCC AGCGTGGCGC TGGACATGAC CGCGCGCAAC
 13261 ATGGAACCGG GCATGTATGC CTCAAACCGG CCGTTTATCA ACCGCCTAAT GGACTACTTG
 13321 CATCGCGGG CGCGCGTGA CCCCGAGTAT TTCACCAATG CCATCTTGAA CCCGCACCTGG
 13381 CTACCGCCCC CTGGTTCTA CACCGGGGGA TTCAAGGTGC CCGAGGGTAA CGATGGATTC
 13441 CTCTGGGACG ACATAGACGA CAGCGTGTG TCCCCGCAAC CGCAGACCCCT GCTAGAGTTG
 13501 CAACAGCGCG AGCAGGCAGA GGCGCGCGT CGAAAAGAAA GCTTCCGCAG GCCAAGCAGC
 13561 TTGTCCGATC TAGGCGCTGC GGCCCCCGCG TCAGATGCTA GTAGCCCATT TCCAAGCTTG
 13621 ATAGGGTCTC TTACCAAGCAC TCGCACCAAC CGCCCGCGCC TGCTGGGCGA GGAGGAGTAC
 13681 CTAACAACT CGCTGCTGCA GCGCGAGCGC GAAAAAAACC TGCCCTCCGGC ATTTCCCAAC
 13741 AACGGGATAG AGAGCCTAGT GGACAAGATG AGTAGATGGA AGACGTACGC GCAGGAGCAC
 13801 AGGGACGTGC CAGGCCCGCG CCCGCCACC CGTCGTAAA GGCACGACCG TCAGCGGGGT
 13861 CTGGTGTGGG AGGACGATGA CTCGGCAGAC GACAGCAGCG TCCTGGATT GGGAGGGAGT
 13921 GGCAACCCGT TTGCGCACCT TCGCCCCAGG CTGGGGAGAA TGTTTTAAAA AAAAAAAAGC
 13981 ATGATGAAA ATAAAAAAACT CACCAAGGCC ATGGCACCGA GCGTTGGTTT TCTTGTATTC
 14041 CCTCTAGTAT GCGGCGCGCG GCGATGTATG AGGAAGGTCC TCCCTCCCTCC TACGAGAGTG
 14101 TGGTGAGCGC GGCGCCAGTG CGCGCGCGC TGGGTTCTCC CTTCGATGCT CCCCTGGACC
 14161 CGCGCTTTGT GCCTCCGCGG TACCTGCGGC CTACCGGGGG GAGAAACAGC ATCCGTIACT
 14221 CTGAGTTGGC ACCCCTATTG GACACCAACCC GTGTGTACCT GGTGGACAAC AAGTCAACGG
 14281 ATGTGGCATC CCTGAACCTAC CAGAACGACCC ACAGCAACTT TCTGACCAACG GTCATTCAA
 14341 ACAATGACTA CAGCCCGGGG GAGGCAAGCA CACAGACCAT CAATCTTGAC GACCGGTGCG
 14401 ACTGGGGCGG CGACCTGAAA ACCATCCTGC ATACCAACAT GCGAAATGTG AACGAGTICA
 14461 TGTTTACCAA TAAGTTTAAG GCGCGGGTGA TGGTGTGCGC CTTGCCTACT AAGGACAATC
 14521 AGGTGGAGCT GAAATACGAG TGGGTGGAGT TCACGCTGCC CGAGGGCAAC TACTCCGAGA
 14581 CCATGACCAT AGACCTTATG AACAACCGCA TCGTGGAGCA CTACCTGAAA GTGGGCAGAC
 14641 AGAACGGGGT TCTGGAAAGC GACATCGGGG TAAAGTTGAA CACCCGCAAC TTCAGACTGG
 14701 GGTTTGACCC CGTCACTGGT CTTGTCTGCA CTGGGGTATA TACAAACGAA GCCTTCCATC
 14761 CAGACATCAT TTGCTGCCA GGATGCGGGG TGGACTTCAC CCACAGCCGC CTGAGCAACT
 14821 TGTTGGGCC CCGCAAGCGG CAACCCCTTC AGGAGGGCTT TAGGATCACC TACGATGATC
 14881 TGGGGGGTGG TAACATTCCC GCACTGTTGG ATGTGGACGC CTACCAAGGGC AGCTTGAAG
 14941 ATGACACCGA ACAGGGCGGG GGTGGCGAG GCGGCAGCAA CAGCAGTGGC AGCGGCAGCG
 15001 AAGAGAACTC CAACCGCGCA GCGCGGGCAA TGCAAGCCGT GGAGGACATG AACGATCATG
 15061 CCATTGCGGG CGACACCTTT GCCACACGGG CTGAGGAGAA GCGCGCTGAG GCGGAAGCAG
 15121 CGGGCGAAGC TGCCGCCCCC GCTGCGCAAC CGGAGGTGCA GAAGCCTCAG AAGAAACCGG
 15181 TGATCAAACC CCTGACAGAG GACAGCAAGA AACGAGTTA CAACCTAATA AGCAATGACA
 15241 GCACCTTCAC CCAGTACCGC AGCTGGTACCT TTGCACTAA CTACGGCGAC CTCAGACCCG
 15301 GAATCCGCTC ATGGACCCCTG CTTTGCACTC CTGACGTAAC CTGCGGCTCG GAGCAGGTCT
 15361 ACTGGTGTGTT GCCAGACATG ATGCAAGACCC CGGTGACCTT CCGCTCCACG CGCCAGATCA
 15421 GCAACTTTCC GGTGGGGC GCGGAGCTGT TGCCCGTGCA CTCCAAGAGC TTCTACAAACG
 15481 ACCAGGGCGT CTACTCCCAA CTCACTCGGC AGTTTACCTC TCTGACCCAC GTGTTCAATC
 15541 GCTTTCCCGA GAACCAAGATT TTGGCGCGCC CGCCAGCCCC CACCATCACC ACCGTCAGTG
 15601 AAAACGTTCC TGCTCTCACCA GATCACGGGA CGCTACCGCT GCGCAACAGC ATCGGAGGAG
 15661 TCCAGCGAGT GACCATTACT GACGCCAGAC GCGCACCTG CCCCTACGTT TACAAGGCC
 15721 TGGGCATAGT CTCGGCGCGC GTCTTATCGA GCGCACTTT TTGAGCAAGC ATGTCCATCC
 15781 TTATATCGCC CAGCAATAAC ACAGGCTGGG GCCTGCGCTT CCAAGCAAG ATGTTTGGCG
 15841 GGGCCAAGAA GCGCTCCGAC CAACACCCAG TGCGCGTGCG CGGGCACTAC CGCGCGCCCT
 15901 GGGGCGCGCA CAAACCGCGC CGCACTGGGC GCACCAACGT CGATGACGCC ATCGACGCC
 15961 TGGTGGAGGA GGCAGCGCAAC TACACGCCA CGCGCCACCC AGTGTCCACA GTGGACGCC
 16021 CCATTCAAGAC CGTGGTGCAC CGAGCCCGC GCTATGCTAA AATGAAGAGA CGGGCGAGGC
 16081 GCGTAGCACG TCGCCACCGC CGCCGACCCG GCACTGCCGC CCAACCGCGC CGGGCGGCC

FIGURE 22
(SHEET 5)

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16141 TGCTTAACCG CGCACGGTCGC ACCGGCCGAC GGGCGGCCAT GCGGGCCGCT CGAAGGCTGG
 16201 CCGCGGGTAT TGTCACTGTG CCCCCCAGGT CCAGGGCAGC AGCGGCCGCC GCAGCAGCCG
 16261 CGGCCATTAG TGCTATGACT CAGGGTCGCA GGGGCAACGT GTATTGGGTG CGCGACTCGG
 16321 TTAGCGGCCT GCGCGTGCCT GTGCGCACCC GCCCCCGCG CAACTAGATT GCAAGAAAAA
 16381 ACTACTTAGA CTCGTACTGT TGTATGTATC CAGCGCGGC GGCGCGCAAC GAAGCTATGT
 16441 CCAAGCGCAA AATCAAAGAA GAGATGCTCC AGGTCACTGC GCGGGAGATC TATGGCCCC
 16501 CGAAGAAGGA AGAGCAGGAT TACAAGCCCC GAAAGCTAAA GCGGGTCAAA AAGAAAAAGA
 16561 AAGATGATGA TGATGAACCT GACGACGAGG TGGAACTGCT GCACGCTACC GCGCCAGGC
 16621 GACGGGTACA GTGGAAAGGT CGACGCGTAA AACGTGTTT GCGACCCGGC ACCACCGTAG
 16681 TCTTACGCC CGGTGAGCGC TCCACCCGCA CCTACAAGCG CGTGTTATGAT GAGGTGTACG
 16741 GCGACGAGGA CCTGCTTGAG CAGGCCAACG AGCGCCTCGG GGAGTTGCCC TACGGAAAGC
 16801 GGCATAAGGA CATGCTGGCG TTGCGCTGG ACGAGGGCAA CCCAACACCT AGCCTAAAGC
 16861 CCGTAACACT GCAGCAGGTG CTGCCCGCGC TTGCAACGTC CGAAGAAAAG CGCGGCCCTAA
 16921 AGCGCGAGTC TGGTGACTTG GCACCCACCG TGCAGCTGAT GGTACCCAAG CGCCAGCGAC
 16981 TGGAAAGATGT CTTGGAAAAA ATGACCGTGG AACCTGGCT GGAGCCCGAG GTCCCGCTG
 17041 GCCCAATCAA GCAGGTGGCG CGGGGACTGG CGTGCGAGAC CGTGGACGTT CAGATACCA
 17101 CTACCAAGTAG CACCAAGTATT GCCACCGCCA CAGAGGGCAT GGAGACACAA ACGTCCCCGG
 17161 TTGCCTCAGC GGTGGGGAT GCCGCGGTGC AGGCGGTGCG TGCGGCCGCG TCCAAGACCT
 17221 CTACGGAGGT GCAAACGGAC CCGTGGATGT TTGCGCTTC AGCCCCCGG CGCCCGCGCG
 17281 GTTCGAGGAA GTACGGCGCC GCAAGCGCGC TACTGCCCCA ATATGCCCTA CATCCTTCCA
 17341 TTGCGCTTAC CCCCCGGCTAT CGTGGCTACA CCTACCGCCC CAGAAGACGA GCAACTACCC
 17401 GACGCCGAAC CACCAACTGGA ACCCGCCGCC GCGTCGCCG TCGCCAGCCC GTGCTGGCCC
 17461 CGATTTCCGT GCGCAGGGTG GCTCGCGAAG GAGGCAGGAC CCTGGTGCTG CCAACAGCGC
 17521 GCTACCAACCC CAGCATCGTT TAAAAGCCGG TCTTGTGGT TCTTGCAGAT ATGGCCCTCA
 17581 CCTGCGCCCT CCGITTTCCCG GTGCGGGGAT TCCGAGGAAG AATGCACCGT AGGAGGGCA
 17641 TGGCCGGCCA CGGCCTGACG GGCGGCATGC GTCGTGCGCA CCACCGGGCG CGGCGCGCGT
 17701 CGCACCGTCG CATGCGCGGC GGTATCTGC CCCTCCTTAT TCCACTGATC GCGCGGGCGA
 17761 TTGGCGCCGT GCGCGGAATT GCATCGTGG CCGTGCAGGC GCAGAGACAC TGATTAAGAA
 17821 CAAGTTGCAT GTGGAAAAAT CAAAATAAAA AGTCTGGACT CTCACGCTCG TTGGTCTCG
 17881 TAACTATTTT TAGAATGGA AGACATCAAC TTTGCGTCTC TGGCCCCGCG ACACGGCTCG
 17941 CGCCCCGTTCA TGGGAAACTG GCAAGATATC GGCACCAAGCA ATATGAGCGG TGGCGCTTC
 18001 AGCTGGGGCT CGCTGTGGAG CGGCATTAAA AATTCGGTT CCACCGTTAA GAACTATGGC
 18061 AGCAAGGCT GGAACACCGAC CACAGGCCAG ATGCTGAGGG ATAAGTTGAA AGAGCAAAAT
 18121 TTCCAACAAA AGGTGGTAGA TGGCCTGGCC TCTGGCATTA GCGGGGGTGGT GGACCTGGCC
 18181 AACCAAGCGAG TGCAAAATAA GATTAACAGT AAGCTTGTATC CCCGCCCTCC CGTAGAGGAG
 18241 CCTCCACCGG CGGTGGAGAC AGTGTCTCCA GAGGGCGTG GCGAAAAGCG TCCGCGCCCC
 18301 GACAGGGAAAG AAACCTCTGGT GACGCAAATA GACGAGCCTC CCTCGTACGA GGAGGCACTA
 18361 AAGCAAGGGC TGCCCCACCAC CGGTCCCCATC GCGCCCATGG CTACCGGAGT GCTGGGCCAG
 18421 CACACACCCG TAACGCTGGA CCTGCGCTCCC CCCGCCGACA CCCAGCAGAA ACCTGTGCTG
 18481 CCAGGCCCGA CGGCCGTTGT TGTAAACCGT CCTAGCCGCG CGTCCCTGCG CGCGCCGCC
 18541 AGCGGTCCGC GATCGTTGCG GCGCGTAGCC AGTGGCAACT GGCAAAGCAC ACTGAACAGC
 18601 ATCGTGGGTC TGGGGGTGCA ATCCCTGAAG CGCCGACGAT GCTCTGAAT AGCTAACGTG
 18661 TCGTATGTT GTCATGTATG CGTCCATGTC GCCGCCAGAG GAGCTGCTGA GCCGCCGCC
 18721 GCCCCCTTC CAAGATGGCT ACCCCCTTCGA TGATGCCGCA GTGGTCTTAC ATGCACATCT
 18781 CGGGCCAGGA CGCCTCGGAG TACCTGAGCC CGGGGCTGGT GCAGTTTGCC CGCGCCACCG
 18841 AGACGTACTT CAGCCTGAAT AACAAAGTTA GAAACCCAC GGTGGCGCCT ACGCACGACG
 18901 TGACCACAGA CGGGTCCCGAG CGTTTGACGC TGCGGTTCAT CCTCTGTGGAC CGTGAGGATA
 18961 CTGCGTACTC GTACAAGGCG CGGTTCACCC TAGCTGTGGG TGATAACCGT GTGCTGGACA
 19021 TGGCTTCCAC GTACTTGAC ATCCGCGGCC TGCTGGACAG GGCCCTACT TTTAACCCCT
 19081 ACTCTGGCAC TGCCTACAAC GCGCTGGCTC CCAAGGGTGC CCCAAATCCT TGCGAACATGG
 19141 ATGAAGCTGC TACTGCTCTT GAATAAAACC TAGAAGAAGA GGACGATGAC AACGAAGACG
 19201 AAGTAGACGA GCAAGCTGAG CAGCAAAAGA CTCACGTATT TGGCAGGGCG CCTTATTCTG
 19261 GTATAAAATAT TACAAAGGAG GTTATTCAA TAGGTGTGCA AGGTCAAACCA CCTAAATATG
 19321 CCGATAAAAC ATTTCAACCT GAAACCTCAA TAGGAGAATC TCAAGTGGTAC GAAACTGAAA
 19381 TTAATCATGC AGCTGGGAGA GTCCCTAAAG AGACTACCCC AATGAAACCA TGTACGGTT
 19441 CATATGCAAACCCACAAAT GAAAATGGAG GGCAAGGCAT TCTTGTAAAG CAACAAATG
 19501 GAAAGCTAGA AAGTCAAGTG GAAATGCAAT TTTCTCAAC TACTGAGGCG ACCGCAGGCA

19561 ATGGTGATAA CTTGACTCCT AAAGTGGTAT TGTACAGTGA AGATGTAGAT ATAGAAACCC
 19621 CAGACACTCA TATTTCTTAC ATGCCCACTA TTAAGGAAGG TAACTCACGA GAACTAATGG
 19681 GCCAACAAATC TATGCCCAAC AGGCCTAATT ACATTGCTTT TAGGGACAAT TTTATTGGTC
 19741 TAATGTATTA CAACAGCACG GTATAATATGG GTGTTCTGGC GGGCCAAGCA TCGCAGTTGA
 19801 ATGCTGTTGT AGATTTGCAA GACAGAAACA CAGAGCTTTC ATACCAGCTT TTGCTTGATT
 19861 CCATTGGTGA TAGAACCAAGG TACTTTCTA TGTGGAATCA GGCTGTTGAC AGCTATGATC
 19921 CAGATGTTAG AATTATTGAA AATCATGGAA CTGAAGATGA ACTTCCAAAT TACTGCTTTC
 19981 CACTGGGAGG TGTGATTAAT ACAGAGACTC TTACCAAGGT AAAACCTAAA ACAGGTCAAGG
 20041 AAAATGGATG GGAAAAAAGAT GCTACAGAAAT TTTCAGATAA AAATGAAATA AGAGTTGAA
 20101 ATAATTTTGC CATGAAATC AATCTAAATG CCAACCTGTG GAGAAATTTC CTGTAACCTCA
 20161 ACATAGCGCT GTATTTGCC GACAAGCTAA AGTACAGTCC TTCCAACGTA AAAATTCTG
 20221 ATAACCCAAA CACCTACGAC TACATGAACA AGCGAGTGGT GGCTCCCAGG TTAGTGGACT
 20281 GCTACATTA CTTGGAGCA CGCTGGTCCC TTGACTATAT GGACAACGTC AACCCATTTA
 20341 ACCACCACCG CAATGCTGGC CTGCGCTACC GCTCAATGTT GCTGGGCAAT GGTCGCTATG
 20401 TGCCCTTCCA CATCCAGGTG CCTCAGAAGT TCTTTGCCAT TAAAAACCTC CTTCTCCTGC
 20461 CGGGCTCAT A CACCTACGAG TGGAACTTCA GGAAGGATGT TAACATGGTT CTGCAGAGCT
 20521 CCCTAGGAAA TGACCTAAGG GTTGACGGAG CCAGCATTAA GTTGTATAGC ATTGCTTCTT
 20581 ACGCCACCTT CTTCCCCATG GCCAACAAACA CGCCTCCAC GTTGTAGGCA ATGCTTAGAA
 20641 ACGACACCAA CGACAGTCC TTAAACGACT ATCTCTCCGC CGCAACATG CTCTACCTA
 20701 TACCCGCAA CGCTACCAAC GTGCCCCATAT CCATCCCCTC CGCCTACTGG GCGGCTTCC
 20761 GCGGCTGGC CTTCACGGC CTTAAGACTA AGGAAACCCCC ATCACTGGGC TCGGGCTACG
 20821 ACCCTTATTA CACCTACTCT GGCTCTATAC CCTACCTAGA TGAACCTTT TACCTCAACC
 20881 ACACCTTAA GAAGGTGGCC ATTACCTTTG ACTCTCTGT CAGCTGGCCT GGCAATGACC
 20941 GCCTGCTTAC CCCAACGAG TTGAAATTAA AGCCTGCTAGT TGACGGGGAG GTTACAACG
 21001 TTGCCCAGTG TAACATGACC AAAGACTGGT TCCCTGGTACA AATGCTAGCT AACTACAACA
 21061 TTGGCTTACCA GGGCTTCTAT ATCCCAGAGA GCTACAGGA CGCATGTAC TCCTTCTTTA
 21121 GAAAATCCA GCCCATGAGC CGTCAGGTGG TGGATGATAC TAAATACAAG GACTACCAAC
 21181 AGGTGGGCAT CCTACACCAA CACAACAAT CTGGATTGT TGGCTACCTT GCCCCCACC
 21241 TGCCTGAAAG ACAGGCCTAC CTCGCTAACT TCCCCTATCC GCTTATAGGC AAGACCCAG
 21301 TTGACAGCAT TACCCAGAAA AAGTTCTTT GCGATCGCAC CCTTGGCGC ATCCCATTCT
 21361 CCAGTAACCT TATGTCCATG GGCGCACTCA CAGACCTGGG CCAAAACCTT CTCTACGCC
 21421 ACTCCGCCA CGCGCTAGAC ATGACTTTTG AGGTGGATCC CATGGACGAG CCCACCCCTC
 21481 TTATGTTT GTTGAAGTC TTGACGTGG TCCGTGTGCA CGGGCCGCAC CGCGGGTCA
 21541 TCGAAACCGT GTACCTGCGC ACGCCCTCT CGGCCGCAA CGCCACAAACA TAAAGAAGCA
 21601 AGCAACATCA ACAACAGCTG CCGCCATGGG CTCCAGTGG CAGGAACCTGA AAGCCATTGT
 21661 CAAAGATCTT GGTGTGGGC CATATTTTTT GGGCACCTAT GACAAGCGCT TTCCAGGCTT
 21721 TGTCTCCA CACAAGCTCG CCTGCGCCAT AGTCAATACG GCCGGTGGCG AGACTGGGG
 21781 CGTACACTGG ATGGCTTGTG CCTGGAACCC GCACTAAAA ACATGCTACC TCTTTGAGCC
 21841 CTTGGCTTT TCTGACCAGC GACTCAAGCA GTTGTACAG TTTGAGTACG AGTCACTCCT
 21901 GCGCCGTAGC GCCATTGCTT CTTCCCCCGA CGCTGTATA ACAGCTGGAAA AGTCCACCCA
 21961 AAGCGTACAG GGGCCAACCT CGGCCGCCTG TGGACTATTG TGTGCTATGT TTCTCCACGC
 22021 CTTTGCAAC TGGCCCCAAA CTCCCATGGA TCACAACCCCC ACCATGAACCC TTATTACCGG
 22081 GGTACCCAAAC TCCATGCTCA ACAGTCCCCA GGTACAGCCC ACCCTGCGTC GCAACCCAGGA
 22141 ACAGCTCTAC AGCTTCTGG AGCGCCACTC GCCCTACTTC CGCAGCCACCA GTGCGCAGAT
 22201 TAGGAGCGCC ACTTTTTTG TGTACTTGAA AAACATGTAA AAATAATGTAA CTAGAGACAC
 22261 TTCAATAAA GGCAAATGCT TTATTTGTA CACTCTCGGG TGATTATTTA CCCCCACCC
 22321 TGCGCTCTGC GCCGTTAAA AATCAAAGGG GTTCTGCGCG GCATCGCTAT GCGCCACTGG
 22381 CAGGGACACG TTGCGATACT GGTGTTTAGT GCTCCACTTA AACTCAGGGCA CAACCACCG
 22441 CGGCAGCTCG GTGAAGTTT CACTCCACAG GCTGCGCACC ATCACCAACG CGTTTACGAG
 22501 GTCGGGCGCC GATATCTTGA AGTCGCGATT GGGGCTCCCG CCTGCGCGC GCGAGTTGCG
 22561 ATACACAGGG TTGCGACACT GGAACACTAT CAGCGCCGGG TGGTGCACGC TGGCCAGCAC
 22621 GCTCTTGTG GAGATCGAT CGCGCTCCAG GTCCCTCCGGG TTGCTCAGGG CGAACGGAGT
 22681 CAACTTTGGT AGCTGCTTC CCAAAAGGG CGCGTGGCCA GGCTTTGAGT TGCACTCGCA
 22741 CCGTAGTGGC ATCAAAGGT GACCGTGGCC GGTCTGGGG TTAGGATACA GCGCTCGCAT
 22801 AAAAGCCTTG ATCTGCTTAA AAGCCACCTG AGCCTTTGCG CCTTCAGAGA AGAACATGCC
 22861 GCAAGACTTG CGGGAAAAT GATTGGCCGG ACAGGGCGCG TCGTGCACGC AGCACCTTGC
 22921 GTCGGTGTG GAGATCTGCA CCACATTCTG GCCCCACCGG TTCTTCACGA TCTTGGCCTT

FIGURE 22
(SHEET 7)

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22981 GCTAGACTGC TCCTTCAGCG CGCGCTGCC 6 GTTTCGCTC GTCACATCCA TTTCAATCAC
 23041 GTGCTCTTA TTTATCATAA TGCTTCCGTG TAGACACTTA AGCTCGCC 7 TT CGATCTCAGC
 23101 GCAGCGGTGC AGCCACAACG CGCAGCCCGT GGGCTCGTGA TGCTTGTAGG TCACCTCTGC
 23161 AAACGACTGC AGGTACGCCT GCAGGAATCG CCCCATCATC GTCACAAAGG TCTTGTGCT
 23221 GGTGAAGGTC AGCTGCAACC CGCGGTGCTC CTCGTTAGC CAGGTCTTGC ATACGGCCGC
 23281 CAGAGCTTCC ACTTGGTCAG GCAGTAGTTT GAAGTTCGCC TTTAGATCGT TATCCACGTG
 23341 GTACTTGTC ATCAGCGCAG GCGCAGCCTC CATGCCCTTC TCCCACGCAG ACACGATCGG
 23401 CACACTCAGC GGGTTCATCA CGCTAATTTC ACTTTCCGCT TCGCTGGGCT CTTCCCTCTTC
 23461 CTCTTGCGTC CGCATACCCAC GCGCCACTGG GTCTGCTTCA TTCA CGCCGCC GCACTGTGCG
 23521 CTTACCTCTT TTGCCATGCT TGATTAGCAC CGGTGGGTTG CTGAAACCCA CCATTTGTAG
 23581 CGCCACATCT TCTCTTCTT CCTCGCTGTC CACGATTACC TCTGGTGTATG GCGGGCGCTC
 23641 GGGCTTGGGA GAAGGGCGCT TCTTTTCTT CTTGGCGCA ATGGCAAAT CCGCCGCGA
 23701 GGTCGATGGC CGCGGGCTGG GTGTGCGCG CACCAGCGCG TCTTGTGATG AGTCTTCCTC
 23761 GTCCCTCGGAC TCGATAACGCC GCCTCATCCG CTTTTTGGG GCGGCCCCGGG GAGGCGGCGG
 23821 CGACGGGGAC GGGGACGACA CGTCCTCCAT GTTGGGGGG ACGCGCGCCG CACCGCGTCC
 23881 GCGCTCGGGG GTGGTTTCGC GCTGCTCTC TTCCCAGCTG GCCATTCTTCT TCTCCTATAG
 23941 GCAGAAAAAG ATCATGGAGT CAGTCGAGAA GAAGGACAGC CTAACCGCCC CCTCTGAGTT
 24001 CGCCACCACCC GCCTCACCG ATGCCGCCAA CGCGCTTAC ACCCTCCCCG TCGAGGCACC
 24061 CCCGCTTGAG GAGGAGGAAG TGATTATCGA GCAGGACCCA GTTTTGTA GCGAACAGCA
 24121 CGAGGACCGC TCAGTACCAA CAGAGGATA AAAGCAAGAC CAGGACAAAG CAGAGGCAAA
 24181 CGAGGAACAA GTCGGGGGGG GGGAGGAAAG GCATGGCGAC TACCTAGATG TGGGAGACGA
 24241 CGTGTGTTG AAGCATCTGC AGCGCCAGTG CGCATTATC TGCGACGCGT TGCAAGAGCG
 24301 CAGCGATGTG CCCCTCGCCA TAGCGGATGT CAGCCTTGCC TACGAACGCC ACCTATTCTC
 24361 ACCCGCGCTA CCCCCCAAAC GCGAACAAAA CGGCACATGC GAGCCCAACC CGCGCTCAA
 24421 CTTCTACCCC GTATTGCGC TGCCAGAGGT GCTTGCACC TATCACATCT TTTTCAAAA
 24481 CTGCAAGATA CCCCCTATCCT GCCGTGCCA CCGCAGCGA GCGGACAAGC AGCTGGCCTT
 24541 GCGGAGGGC GCTGTCATAC CTGATATCGC CTCGCTAAC GAAGTGCCTA AAATCTTGA
 24601 GGGTCTTGA CGCGACGAGA AGCGCGCGC AACGCTCTG CAACAGGAAA ACAGCGAAAA
 24661 TGAAAGTCAC TCTGGAGTGT TGGTGGAACT CGAGGGTGC AACGCGCGCC TAGCGTACT
 24721 AAAACGAGC ATCGAGGTCA CCCACTTTGC CTACCCCGCA CTTAACCTAC CCCCCAAGGT
 24781 CATGAGCACA GTCATGAGTG AGCTGATCGT GCGCCGTGCG CAGCCCCCTGG AGAGGGATGC
 24841 AAATTGCAA GAACAAACAG AGGAGGGCCT ACCCGCAGTT GGGCAGCAGC AGCTAGCGCG
 24901 CTGGCTCAA ACGCCGAGC CTGCCGACTT GGAGGAGCGA CGCAAACCTAA TGATGGCCG
 24961 AGTGCTCGTT ACCGTGGAGC TTGAGTGCAT GCAGCGGTT TTTGCTGACC CGGAGATGCA
 25021 GCGCAAGCTA GAGGAAACAT TGCACATACAC CTTTCGACAG GGCTACGTAC GCCAGGCTG
 25081 CAAGATCTCC AACGTGGAGC TCTGCAACCT GGTCTCCTAC CTGGAAATT TGACGAAAA
 25141 CCGCCTGGG CAAACGTGC TTCATTCCAC GCTCAAGGG CAGGCGCGCC GCGACTACGT
 25201 CCGCGACTGC GTTTACTTAT TTCTATGCTA CACCTGGCAG ACCGCCATGG GCGTTGGCA
 25261 GCAGTGCTTG GAGGAGTGCA ACCTCAAGGA GCTGCAGAA CTGCTAAAGC AAAACTTGA
 25321 GGACCTATGG ACGGCCCTCA ACGAGCGCTC CGTGGCGCG CACCTGGCGG ACATCATTT
 25381 CCCCAGACG CTGCTTAAAA CCCTGCAACA GGGTCTGCCA GACTTCACCA GTCAAAGCAT
 25441 GTTGCAGAAC TTTAGGAACT TTATCCTAGA GCGCTCAGGA ATCTTGGCCG CCACCTGCTG
 25501 TGCACTTCCT AGCGACTTTG TGCCCATTAA GTACCGCGA TGCCCTCCGC CGCTTTGGGG
 25561 CCACTGCTAC CTTCTGCAGC TAGCCAACTA CCTTGCTTAC CACTCTGACA TAATGGAAGA
 25621 CGTGAGCGGT GACGGTCTAC TGGAGTGTCA CTGTCGCTGC AACCTATGCA CCCCCCACCG
 25681 CTCCCTGGTT TGCAATTGCG AGCTGTTAA CGAAAGTCAA ATTATCGGTA CCTTTGAGCT
 25741 GCAGGGTCCC TCGCCGTACG AAAAGTCCGC GGCTCCGGGG TTGAAACTCA CTCCGGGGCT
 25801 GTGGACGTCG GCTTACCTTC GCAAATTGTT ACCTGAGGAC TACCAACGCC ACGAGATTAG
 25861 GTTCTACGAA GACCAATCCC GCGCGCCAA TGCGGAGCTT ACCGGCCTGCG TCATTACCCA
 25921 GGGCCACATT CTGCGCAAT TGCAAGGCC CAACAAAGCC CGCCAAAGAGT TTCTGCTACG
 25981 AAAGGGACGG GGGGTTTACT TGGACCCCCA GTCCGGCGAG GAGCTCAACC CAATCCCCC
 26041 GCCGCCGCGAG CCCTATCAGC AGCAGCGCG GGGCTTGC TCCCAAGGATG GCACCCAAAA
 26101 AGAACGCTGCA GCTGCCGCGC CAACCCACGG ACGAGGAGGA ATACTGGGAC AGTCAGGCAG
 26161 AGGAGGTTT GGACGGAGAG GAGGAGGACA TGATGGAAGA CTGGGAGAGC CTAGACGAGG
 26221 AAGCTTCCGA GGTGCAAGAG GTGTCAGACG AAACACCGTC ACCCTCGGTC GCATTCCCC
 26281 CGCCGGCGCC CCAGAAATCG GCAACCGGTT CCAGCATGGC TACAACCTCC GCTCCTCAGG
 26341 CGCCGCCGGC ACTGCCGTT CGCCGACCCA ACCGTAGATG GGACACCACT GGAACCAGGG

26401 CCGGTAAGTC CAAGCAGCCG CGGCCGTAG CCCAAGAGCA ACAACAGCGC CAAGGCTACC
 26461 GCTCATGGCG CGGGCACAAAG AACGCCATAG TTGCTTGCTT GCAAGACTGT GGGGGCAACA
 26521 TCTCCCTTCGC CCGCCGCTTT CTTCTCTACC ATCACGGCGT GGCCCTTCCCC CGTAACATCC
 26581 TGCAATTACTA CCGTCATCTC TACAGCCCAT ACTGCACCGG CGGCAGCGC AGCGGCAGCA
 26641 ACAGCAGCGG CCACACAGAA GCAAAGGGCGA CGGATAGCA AGACTCTGAC AAAGCCCAAG
 26701 AAATCCACAG CGGCGGCAGC AGCAGGGAGGA GGAGCGCTGC GTCTGGCGCC CAACGAACCC
 26761 GTATCGACCC GCGAGCTTAG AAACAGGATT TTTCCCACTC TGTATGCTAT ATTTCAACAG
 26821 AGCAGGGGCC AAGAACAAAG GCTAAAATA AAAAACAGGT CTCTGCGATC CCTCACCCGC
 26881 AGCTGCCCTGT ATCACAAAAG CGAAGATCAG CTTCGCGCA CGCTGGAAGA CGCGGAGGCT
 26941 CTCTTCAGTA AATACTGCGC GCTGACTCTT AAGGACTAGT TTGCGGCCCT TTCTCAAATT
 27001 TAAGCGCAA AACTACGTCA TCTCCAGCGG CCACACCCCG CGCCAGCACC TGTCGTCAAG
 27061 GCCATTATGA GCAAGGAAAT TCCCACGCCC TACATGTGGA GTTACCGAGCC ACAAAATGGGA
 27121 CTTGCGGCTG GAGCTGCCA AGACTACTCA ACCCGAATAA ACTACATGAG CGCGGGACCC
 27181 CACATGATAT CCCGGGTCAA CGGAATCCGC GCCCACCGAA ACCGAATTCT CTTGGAACAG
 27241 GCGGCTATTA CCACACACC TCGTAATAAC CTTAACCCCC GTAGTTGGCC CGCTGCCCTG
 27301 GTGTACCCAGG AAAGTCCCAGC TCCCACCACT GTGGTACTTC CCAGAGACGC CCAGGCCGAA
 27361 GTTCAGATGA CTAACTCAGG GCGCGAGCT GCGGGCGCT TTGTCACAG GGTGCGGTGCG
 27421 CCCGGGCAGG GTATAACTCA CTCGACAAATC AGAGGGCGAG GTATTCACTC CAACGACGAG
 27481 TCCTGTGAGCT CCTCGCTTGG TCTCCGTCCG GACGGGACAT TTCACTCGG CGGCGCCGGC
 27541 CGTCCTTCAT TCACGCCCTG TCAGGCAATC CTAACTCTGC AGACCTCGTC CTCTGAGCCG
 27601 CGCTCTGGAG GCATTGGAAC TCTGCAATT ATTGAGGAGT TTGTCATCT GGTCTACTTT
 27661 AACCCCTTCT CGGGACCTCC CGGCCACTAT CGGATCAAT TTATTCTAA CTTTGACCGG
 27721 GTAAAGGACT CGGGGACGG CTACGACTGA TAATTAAGTG GAGAGGCAGA GCAACTGCGC
 27781 CTGAAACACC TGGTCCACTG TCGCCGCCAC AAGTGTGTTG CCGCGACTC CGGTGAGTTT
 27841 TGCTACTTGT AATGGCCGA GGATCATATC GAGGATCTTT GTGCCATCT CTGTGCTGAG
 27901 TATAATAAAAT ACAGAAATTAA AAATATACTG GGGCTCTAT CGCCATCCTG TAAACGCCAC
 27961 CGTCTTCACC CGCCCAAGCA AACCAAGGG AACCTTACCT GGTACTTTITA ACATCTCTCC
 28021 CTCTGTGATT TACAACAGTT TCAACCCAGA CGGAGTGGAGT CTACGAGAGA ACCTCTCCGA
 28081 GCTCAGCTAC TCCATCAGAA AAAACACCAC CCTCCTTACCG TGCCGGGAAC GTACCCCTAA
 28141 TTAAAAGTCA GGCTTCCCTGG ATGTCAGCAT CTGACTTTGG CCAGCACCTG TCCCAGGGAT
 28201 TTGTTCCAGT CCAACTACAG CGACCCACCC TAACAGAGAT GACCAACACA ACCAACGCGG
 28261 CGCCGCTAC CGGACTTACA TCTACCACAA ATACACCCCA AGTTTCTGCC TTTGTCAATA
 28321 ACTGGGATAA CTTGGGCATG TGGTGGTTCT CCATAGCGCT TATGTTTGTA TGCCCTTATTA
 28381 TTATGTGGCT CATCTGCTGC CTAAAGCGCA AACGCGCCCG ACCACCCATC TATAGTCCA
 28441 TCATTGTGCT ACACCCAAAC ATGATGGAA TCCATAGATT GGACGGACTG AAACACATGT
 28501 TCTTTCTCT TACAGTATGA TAAATGAGA TTAATTAAGG ATTTCCTGTC CAGTTTATTC
 28561 AGCAGCACCT CCTTGCCCTC CTCCCAGCTC TGGTATGCA GTTCCCTCCT GGCTGAAAC
 28621 TTCTCCACA ATCTAAATGG ATGTCAGTT TCCCTCTGTT CCTGTCACAT CGCACCCACT
 28681 ATCTTCATGT TGTTGCAGAT GAAGCGCGCA AGACCGCTG AAGATACCTT CAACCCCGTG
 28741 TATCCATATG ACACGGAAAC CGGTCTCTCA ACTGTGCCCTT TTCTTACTCC TCCCTTGTG
 28801 TCCCCCAATG GGTTCAAGA GAGTCCCCCT GGGGTACTCT CTTTGCGCCT ATCCGAACCT
 28861 CTAGTTACTT CCAATGGCAT CCTTGGCGCTC AAAATGGGA ACAGGCCCTC TCTGGACGAG
 28921 GCCGGCAACC TTACCTCCCA AAATGTAACC ACTGTGAGCC CACCTCTCAA AAAAACCAAG
 28981 TCAAAACATAA ACCTGGAAAT ATCTGCACCC CTCACAGTTA CCTCAGAAGC CCTAACTGTG
 29041 GCTGCCGCCG CACCTCTAAT GTCGCGGGC AACACACTCA CCATGCAATC ACAGGCCCG
 29101 CTAACCGTGC ACGACTCCAA ACTTAGCATT CGCACCCAG GACCCCTCAC AGTGTAGAA
 29161 GGAAAGCTAG CCTGCAAAC ATCAGGGCCC CTCACCCCA CGGATAGCAG TACCCCTACT
 29221 ATCACTGCTC CACCCCTCT AACTACTGCCC ACTGGTAGCT TGGGCATTGA CTTGAAAGAG
 29281 CCCATTATAA CACAAAATGG AAAACTAGGA CTAAAGTAGC GGGCTCCTTT GCATGTAACA
 29341 GACGACCTAA ACACTTGAC CGTAGCAACT GGTCCAGGTG TGAATTAA TAATACTTCC
 29401 TTGCAAACCA AAGTTACTGG AGCCTGGGT TTGATTCAC AAGGCAATAT GCAACTTAAT
 29461 GTAGCAGGGAG GACTAAGGAT TGATTCTCAA AACAGACGCC TTATACTTGA TGTTAGTTAT
 29521 CCGTTTGATG CTCAAAACCA ACTAAATCTA AGACTAGGAC AGGGCCCTCT TTTTATAAAC
 29581 TCAGCCACA ACTGGATAT TAACTACAA ACAGGCCCTT ACTTGTGTTAC AGCTTCAACAC
 29641 AATTCCAAAA AGCTTGAGGT TAACCTAACG ACTGCCAAGG GGTGATGTT TGACGCTAC
 29701 GCCATAGCCA TTAATGCAGG AGATGGGCTT GAATTGGTT CACCTAATGC ACCAAACACA
 29761 AATCCCCCTCA AAACAAAAAT TGGCCATGGC CTAGAATTG ATTCAAACAA GGCTATGGTT

FIGURE 22
(SHEET 9)

kd1

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42k6

29821 CCTAAACTAG GAACTGGCCT TAGTTTGAC AGCACAGGTG CCATTACAGT AGGAAACAAA
 29881 AATAATGATA AGCTAACTTT GTGGACCACA CCAGCTCCAT CTCTTAACGT TAGACTAAAT
 29941 GCAGAGAAAG ATGCTAACT CACTTTGGTC TAAACAAAAT GTGGCAGTC AATACTTGCT
 30001 ACAGTTTCAG TTTTGGCTGT TAAAGGCAGT TTGGCTCCAA TATCTGGAAC AGITCAAAGT
 30061 GCTCATCTTA TTATAAGATT TGACGAAAAT GGAGTGTAC TAAACAATTG CTTCTGGAC
 30121 CCAGAATATT GGAACCTTAG AAATGGAGAT CTTACTGAAG GCACAGCCTA TACAAACGCT
 30181 GTTGGATTTA TGCTTAACCT ATCAGCTTAT CAAAAATCTC ACGGTTAAAC TGCCAAAAGT
 30241 AACATTGTC A GTCAAGTTA CTTAAACGGA GACAAAACTA AACCTGTAAC ACTAACCTT
 30301 ACACTAAACG GTACACAGGA AACAGGAGAC ACAACTCCAA GTGCATACTC TATGTCATTT
 30361 TCATGGGACT GGTCTGGCCA CAACTACATT AATGAAATAT TTGCCACATC CTCTTACACT
 30421 TTTTCATACA TTGCCCAAGA ATAAAGAACG GTTTGTGTTA TGTTTCAACG TGTTTATTTT
 30481 TCAATTGCG AAAATTCAA GTCATTTTC ATTCAAGTAGT ATAGCCCCAC CACCACATAG
 30541 CTTATACAGA TCACCGTACCTTAAATCAAAC TCACAGAACCT TAGTATTCA ACCTGCACC
 30601 TCCCTCCCA CACACAGAT ACACAGTCCT TTCTCCCCGG CTGGCCTTAA AAAGCATCAT
 30661 ATCATGGGTA ACAGACATAT TCTTAGGTGT TATATTCCAC ACGGTTTCCT GTGAGGCCAA
 30721 ACGCTCATCA GTGATATTAA TAAACTCCCC GGGCAGCTCA CTTAAGTTCA TGTCGCTGTC
 30781 CAGCTGCTGA GCCACAGGCT GCTGTCCAACTTGCAGGTTGCTTAAACGGGCG GCGAAGGAGA
 30841 AGTCCACGCC TACATGGGGG TAGAGTCATA ATCGTGCATC AGGATAGGGC GGTGGTGTG
 30901 CAGCAGCGCG CGAATAAAACT GCTGCGCGCG CGCCTCCGTC CTGCAGGAAT ACAACATGGC
 30961 AGTGGTCTCC TCAGCGATGA TTGCGACCGC CGCAGCATA AGGCGCCTTGCCTTCC
 31021 ACAGCAGCGC ACCCTGATCT CACTTAAATC AGCACAGTAA CTGCAAGCACA GCACCACAAAT
 31081 ATTGTTCAAA ATCCCACAGT GCAAGGCCT GTATCCAAAG CTICATGGCGG GGACCAACAGA
 31141 ACCCACGCTGG CCATCATACC ACAAGCGCAG GTAGATTAAG TGCGACCCCC TCATAAACAC
 31201 GCTGGACATA AACATTACCT TTTTGGCAT GTGTAATT ACCACCTCCCC GGTACCATAT
 31261 AAACCTCTGA TTAAACATGG CGCCATCCAC CACCATCCTA AACAGCTGG CCAAAACCTG
 31321 CCCGCCGGT ATACACTGCA GGGAACCGGG ACTGGAACAA TGACAGTGG GAGCCCAGGA
 31381 CTCGTAACCA TGGATCATCA TGCTCGTCAT GATATCAATG TTGGCACACAC ACAGGCACAC
 31441 GTGCATACAC TTCTCTCAGGA TTACAAGCTC CTCCCGCGTT AGAACCCATAT CCCAGGGAAC
 31501 AACCCATTC TGAATCAGCG TAAATCCCC ACTGCAGGGAGAACCTCGCA CGTAACCTCAC
 31561 GTTGTGCATT GTCAAAGTGT TACATTGGG CAGCAGCGGA TGATCCTCCA GTATGGTAGC
 31621 GCGGGTTCT GTCTCAAAAG GAGGTAGACG ATCCCTACTG TACGGAGTGC GCCGAGACAA
 31681 CCGAGATCGT GTTGGTGTGA GTGTCATGCC AAATGGAACG CCGGACGTAG TCATATTCC
 31741 TGAAGCAAA CCAGGTGGCG GCGTACAAA CAGATCTGCG TCTCCGGTCT CGCCGCTTAG
 31801 ATCGCTCTGT GTAGTAGTTG TAGTATATCC ACTCTCTCAA AGCATCCAG CGCCCCCTGG
 31861 CTTCGGGTT TATGAAACT CCTTCATGCG CCGCTGCCCT GATAACATCC ACCACCGCAG
 31921 AATAAGCCAC ACCCAGCCAA CCTACACATT CGTTCTGCGA GTCAACACACGG GGAGGAGCGG
 31981 GAAGAGCTGG AAGAACCATG TTTTTTTTT TATTCCAAAA GATTATCCAA AACCTCAAA
 32041 TGAAGATCTA TTAAGTGAAC GCGCTCCCT CGGTGGCGT GGTCAAACCTC TACAGCAAA
 32101 GAACAGATAA TGGCATTGTG AAGATGTTG ACAATGGCTT CAAAAAGGCA AACGGCCCTC
 32161 ACGTCCAAGT GGACGTAAG GCTAAACCTC TCAGGGTGA TCTCCTCTAT AAACATTCCA
 32221 GCACCTTCAA CCATGCCAA ATAATTCTCA TCTCGCCACC TTCTCAATAT ATCTCTAAC
 32281 AAATCCGAA TATTAAGTCC GGCCATTGTA AAAATCTGCT CGAGAGCGCC CTCCACCTTC
 32341 AGCCTCAAGC AGCGAATCAT GATTGCAAA ATTCAAGGTT CTCACAGACCG TGTATAAGAT
 32401 TCAAAAGCGG AACATTAACA AAAATACCGC GATCCCCTGAG GTCCCTTCGC AGGGCCAGCT
 32461 GAACATAATC GTGCAGGTCT GCACGGACCA GCGCGGCCAC TTCCCGCCCA GGAACCTTGA
 32521 CAAAAGAACCC CACACTGATT ATGACACGGC TACTCGGAGC TATGCTAACCG AGCGTAGGCC
 32581 CGATGTAAGC TTTGTTGCAT GGGCGCGAT ATAAAATGCA AGGTGCTGCT CAAAAAAATCA
 32641 GGCAAAGCCT CGCGCAAAAAA AGAAAGCACA TCGTAGTCAT GCTCATGCG ATAAAGGCAG
 32701 GTAAGCTCCG GAACACCAC AGAAAAAGAC ACCATTTC TCTCAAACAT GTCTCGGGT
 32761 TTCTGCATAA ACACAAAAA AAATAACAAA AAAACATTAA AACATTAGAA GCCTGCTTA
 32821 CAACAGGAAA AACACCCCTT ATAAGCATAA GACGGACTAC GGCCATGCCG GCGTGACCGT
 32881 AAAAAAAACTG GTCACCGTGA TTTAAAAGCA CCACCGACAG CTCTCTCGTC ATGTCGGAG
 32941 TCATAATGTA AGACTCGGTA AACACATCAG GTTGATTCTAT CGGTCTGAG TAAAAAGCAG
 33001 CCGAAATAGC CCGGGGGAAAT ACATACCCGC AGGCCTAGAG ACAACATTAC AGCCCCCATA
 33061 GGAGGTATAA CAAAATTAAT AGGAGAGAAA AACACATAAA CACCTGAAAA ACCCTCTGC
 33121 CTAGGCAAA TAGCACCTC CCGCTCCAGA ACAACATACA GCGCTTCACA GCGGCAGCCT
 33181 AACAGTCAGC CTTACCAAGTA AAAAGAAAA CCTATTAAA AAACACCACT CGACACGGCA

FIGURE 22
(SHEET 10)

33241 CCAGCTCAAT CAGTCACAGT GTAAAAAAAGG GCCAAGTGCA GAGCGAGTAT ATATAGGACT
33301 AAAAAATGAC GTAACGGTTA AAGTCCACAA AAAACACCCA GAAAACCGCA CGCGAACCTA
33361 CGCCCAGAAA CGAAAGCCAA AAAACCCACA ACTTCCTCAA ATCGTCACTT CCGTTTTCCC
33421 ACGTTACGTA ACTTCCCATT TTAAGAAAAC TACAATTCCC AACACATACA AGTTACTCCG
33481 CCCTAAAACC TACGTCAACCC GCCCCGTTCC CACGCCCGC GCCACGTCAC AAACCTCCACC
33541 CCCTCAATTAT CATATTGGCT TCAATCCAAA ATAAGGTATA TTATTGATGA TG

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kd1

FIGURE 22
(SHEET 11)

11

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LOCUS KD3 **34341 bp** **DNA** **SYN** **06-FEB-1999**
DEFINITION KD3
ACCESSION KD3
KEYWORDS
SOURCE Unknown.
ORGANISM Unknown
Unclassified.
REFERENCE 1 (bases 1 to 34341)
AUTHORS Self
JOURNAL Unpublished.
FEATURES Location/Qualifiers
CDS
 1..34341
 /gene="KD3"
 /product="KD3"
BASE COUNT 7951 a 9671 c 9464 g 7255 t
ORIGIN

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1 CATCATCAAT AATATAACCTT ATTTTGGATT GAAGCCAATA TGATAATGAG GGGGTGGAGT
61 TTGTGACGTG CGCGGGGGCG TGGGAACGGG GCGGGGTGACG TAGTAGTGTG GCGGAAGTGT
121 GATGTTGCAA GTGTGGCGGA ACACATGTA GCGACGGATG TGCACAAAGT GACGTTTTTG
181 GTGTGCGCCG GTGTACACAG GAAGTGACAA TTTCCGCGC GTTTTAGGCG GATGTTGTAG
241 TAAATTTGGG CGTAACCGAG TAAGATTTGG CCATTTCGCG GGGAAAACGT AATAAGAGGA
301 AGTAAAATCT GAATAATTTC GTGTTACTCA TAGCGCGTAA TATTTGTCTA GGGCCGCGGG
361 GACTTTGACC GTTTACGTGG AGACTCGCCC AGGTGTTTT CTCAGGTGTT TTCCGCGTTC
421 CGGGTCAAAG TTGGCGTTT ATTATTATAG TCAGCTGACG TGTAGTGTAT TTATACCCGG
481 TGAGTTCCCTC AAGAGGCCAC TCTTGAGTGC CAGCGAGTAG AGTTTTCTCC TCCGAGCCGC
541 TCCGACACCG GGACTGAAAA TGAGACATGA GGTACTGGCT GATAATCTTC CACCTCTAG
601 CCATTTGAA CCACCTACCC TTCACGAAC GTATGATTTA GACGTGACGG CCCCCGAAAGA
661 TCCCAACGAG GAGGCGGTTT CGCAGATTTC TCCCGACTCT GTAATGTTGG CGGTGCAGGA
721 AGGGATTGAC TTACTCACTT TTCCGCCGGC GCCCCGGTTCT CCGGAGCCGC CTCACCTTTC
781 CCGGCAGGCC GAGCAGGCCGG AGCAGAGAGC CTTGGGTCCG GTTTGCCACG AGGCTGGCTT
841 TCCACCCAGT GACGACGAGG ATGAAGAGGG TGAGGAGTTT GTGTTAGATT ATGTGGAGCA
901 CCCCGGGCAC GGTTCAGGT CTTGTCATTA TCACCCGGAGG AATACGGGG ACCCAGATAT
961 TATGTGTTCG CTTTGTATA TGAGGACCTG TGGCATGTT GTCTACAGTA AGTAAAATT
1021 ATGGGCAGTG GGTGATAGAG TGGTGGGTTT GGTGTGGTAA TTTTTTTTTT AATTTTACA
1081 GTTTTGTGGT TTAAAGAATT TTGTATTGTG ATTTTTTTAA AAGGTCTGT GTCTGAACCT
1141 GAGCCTGAGC CCGAGCCAGA ACCGGAGCCT GCAAGACCTA CCCGCCGTCC TAAAATGGCG
1201 CCTGCTATCC TGAGACGCC GACATCACCT GTGTCTAGAG AATGCAATAG TAGTACGGAT
1261 AGCTGTGACT CGGTCTTC TAACACACT CCTGAGATAC ACCCGGTGGT CCCGCTGTGC
1321 CCCATTAAAC CAGTTGCCGT GAGAGTTGGT GGGCGTCGCC AGGCTGTGGA ATGTATCGAG
1381 GACTTGCTTA ACGAGCCTGG GCAACCTTG GACTTGAGCT GTAAACGCC CAGGCCATAA
1441 GGTGTAAACC TGTGATTGCG TGTGTGGTAA ACGCCCTTGT TTGTGAATG AGTTGATGTA
1501 AGTTTAATAA AGGGTGAGAT AATGTTAAC TTGCATGGCG TGTTAAATGG GGCGGGGCTT
1561 AAAGGGTATA TAATGCGCCG TGGGCTAACAT TTGGTACAT CTGACCTCAT GGAGGCTTGG
1621 GAGTGTGTTGG AAGATTTTC TGCTGTGCGT AACTTGCTGG AACAGAGCTC TAACAGTACC
1681 TCTTGGTTT GGAGGTTTCT GTGGGGCTCA TCCCAGGCAA AGTTAGTCTG CAGAATTAAG
1741 GAGGATTACA AGTGGGAATT TGAAGAGCTT TTGAAATCCT GTGGTGTGAGCT GTTTGATCT
1801 TTGAATCTGG GTCAACAGGC GCTTTTCCAA GAGAAGGTCA TCAAGACTTT GGATTTTCC
1861 ACACGGGGC GCGCTGCCG TGCTGTGCT TTTTGAGTT TTATAAAGGA TAAATGGAGC
1921 GAAGAAACCC ATCTGAGCGG GGGGTACCTG CTGGATTTC TGGCCATGCA TCTGTGGAGA
1981 GCGGTTGTGA GACACAAGAA TCGCCTGCTA CTGTTGTCTT CCGTCGCC GGCAGATAATA
2041 CCGACGGAGG AGCAGCAGCA GCAGCAGGAG GAAGCCAGGC GGCGGGGCCA GGAGCAGAGC
2101 CCATGGAACC CGAGAGCCGG CCTGGACCT CGGGAAATGAA TGTGTTACAG GTGGCTGAAC
2161 TGTATCCAGA ACTGAGACGC ATTTGACAA TTACAGAGGA TGGGCAGGGG CTAAAGGGGG
2221 TAAAGAGGGA GCGGGGGGCT TGTGAGGCTA CAGAGGAGGC TAGGAATCTA GCTTTTAGCT
2281 TAATGACCAG ACACCGTCCT GAGTGTATTA CTTTCAACA GATCAAGGAT AATTGCGCTA
2341 ATGAGCTTGA TCTGCTGGCG CAGAAGTATT CCATAGAGCA GCTGACCAC TACTGGCTGC
2401 AGCCAGGGGA TGATTTTGAG GAGGCTATTA GGGTATATGC AAAGGTGGCA CTTAGGCCAG

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2461 ATTGCAAGTA CAAGATCAGC AAACTTGTAA ATATCAGGAA TTGTTGCTAC ATTTCTGGGA
 2521 ACGGGGCCGA GGTGGAGATA GATA CGGAGG ATAGGGTGGC CTTTAGATGT AGCATGATAA
 2581 ATATGTGGCC GGGGGTGCCTT GGCATGGACG GGGTGGTTAT TATGAATGTA AGGTTTACTG
 2641 GCCCCAATT TAGCGGTACG GTTTTCCTGG CCAATACCAA CCTTATCCTA CACGGTGAA
 2701 GCTTCTATGG GTTTAACAAAT ACCTGTGTGG AAGCCTGGAC CGATGTAAGG GTTCGGGCT
 2761 GTGCCTTTA CTGCTGCTGG AAGGGGGTGG TGTCGCCC CAAAAGCAGG GCTTCATTA
 2821 AGAAATGCCT CTTTGAAAGG TGACCTTG GTATCCTGTC TGAGGGTAAC TCCAGGGTGC
 2881 GCCACAATGT GGCCCTCCGAC TGTTGGTTGCT TCATGCTAGT AAAAGCGTG GCTGTGATTA
 2941 AGCATAAACAT GGTATGTGGC AACTGCGAGG ACAGGGCTC TCAGATGCTG ACCTGCTCGG
 3001 ACGGCAACTG TCACCTGCTG AAGACCATTG ACGTAGCCAG CCACCTCTGC AAGGCCTGGC
 3061 CAGTGTGTTGA GCATAACATA CTGACCCGCT GTTCCTGCA TTGGGTTAAC AGGAGGGGG
 3121 TGTTCCCTACC TTACCAATGC AATTGAGTC ACACAAAGAT ATTGCTTGAG CCCGAGAGCA
 3181 TGTCGAAGGT GAACCTGAAAC GGGGTGTTTG ACATGACCAT GAAGATCTGG AAGGTGCTGA
 3241 GGTACCGATGA GACCCGCACC AGGTGCGAGAC CCTGCGAGTG TGCGGGTAAA CATATTAGGA
 3301 ACCAGCCTGT GATGCTGGAT GTGACCCGAGG AGCTGAGGCC CGATCACTTG GTGCTGGCT
 3361 GCACCCGGCG TGAGTTGGC TCTAGCGATG AAGATACAGA TTGAGGTTACT GAAATGTGTG
 3421 GGCCTGGCTT AAGGGTGGGA AAGAATATAT AAGGGGGGG TCTTATGTAG TTTTGTATCT
 3481 GTTTGCAGC AGCCGCCGCC GCCATGAGCA CCAACTCGTT TGATGGAAGC ATTGTGAGCT
 3541 CATATTTGAC AACGCGCATG CCCCCATGGG CGGGGGTGC TCAGAAATGTG ATGGGCTCCA
 3601 GCATTGATGG TCGCCCCGTC CTGCCCCCAA ACTCTACTAC CTTGACCTAC GAGACCGTGT
 3661 CTGGAACGCC GTTGGAGACT GCAGCCTCG CCGCCGCTTC AGCCGCTGCA GCCACCGCCC
 3721 GCGGGATTGT GACTGACTTT GTTTCTGCA GCCCCCTGCA AAGCAGTGCA GCTTCCCGTT
 3781 CATCCGCCCG CGATGACAAG TTGACGGCTC TTTTGGCACA ATTGGATTCT TTGACCCGGG
 3841 AACTTAATGT CGTTTCTCAG CAGCTGTTGG ATCTGCGCA GCAGGTTCT GCCCTGAAGG
 3901 CTTCCCTCCCC TCCCAATGCG GTTTAAAACA TAAATAAAA ACCAGACTCT GTTTGGATT
 3961 GGATCAAGCA AGTGTCTTGC TGTCTTTAT TAGGGTTTT GCGCGCGCGG TAGGCCCCGG
 4021 ACCAGCGGTC TCGGTGCTTG AGGGTCTGT GTATTTTTTC CAGGACGTGG TAAAGGTGAC
 4081 TCTGGATGTT CAGATACATG GGCATAAGGC CGTCTCTGGG TTGGAGGTTAG CACCACTGCA
 4141 GAGCTTCATG CTGGGGGTG GTGTTGTAGA TGATCCAGTC GTAGCAGGAG CGCTGGCGT
 4201 GGTGCCTAAA AATGTCTTTC AGTAGCAAGC TGATTGCCAG GGGCAGGCC TTGGTGTAAAG
 4261 TGTTCACAAA GCGGTTAACG TGGGATGGGT GCATACGTGG GGATATGAGA TGCACTTGG
 4321 ACTGTATTT TAGTTGGCT ATGTTCCAG CCATATCCCT CCGGGGATTC ATGTTGTGCA
 4381 GAACCACAG CACAGTGTAT CCGGTGCACT TGGGAAATT GTCATGTAGC TTAGAAGGAA
 4441 ATGCGTGGAA GAACTGGAG ACGCCCTTGT GACCTCCAAG ATTTCCTCATG CATTGTC
 4501 TAATGATGCC AATGGGCCCA CGGGCGGCCG CCTGGGCGAA GATATTTCTG GGATCACTAA
 4561 CGTCATAGTT GTGTTCCAGG ATGAGATCGT CATAGGCCAT TTTTACAAAG CGCGGGCGGA
 4621 GGGTGCCAGA CTGGGTATA ATGGTTCCAT CCGGCCAGG GCGTAGTTA CCCTCACAGA
 4681 TTTGCATTC CCACGCTTGT AGTTCAAGTG GGGGGATCAT GTCTACCTGC GGGGGATGA
 4741 AGAAAAACGGT TCCGGGGGTA GGGGAGATCA GCTGGGAAGA AAGCAGGTTTC CTGAGCAGCT
 4801 GCGACTTACG GCAGCCGGTG GGGCGTAA TCACACCTAT TACCGGGTGC AACTGGTAGT
 4861 TAAGAGAGCT GCAGCTGCCG TCATCCCTGA CAGGGGGC CACTTCGTAA AGCATGTCCC
 4921 TGACTCGCAT GTTTCCCTG ACCAAATCCG CCAGAAGGCG CTCGCCGCC AGCGATAGCA
 4981 GTTCTTGCAA GGAAGCAAAG TTTTCAACG GTTTGAGACC GTCCGCCGTA GGCATGCTT
 5041 TGAGCGTTG ACCAAGCAGT TCCAGGGCGT CCCACAGCTC GGTACCTGC TCTACGGCAT
 5101 CTCGATCCAG CATATCTCT CGTTTCCGG GTTGGGGCGG CTTTCGTGT ACGGCAGTAG
 5161 TCGGTGCTCG TCCAGACGGG CCAGGGTCA GTCTTCCAC GGGCGCAGGG TCCTCGTCAG
 5221 CGTAGTCTGG GTCA CGGTGA AGGGGTGGC TCGGGCTGCG GCGCTGGCCA GGGTGGCTT
 5281 GAGGCTGGTC CTGCTGGTGC TGAAGCGCTG CCGGTCTCG CCTCGCGCGT CGGCCAGGTA
 5341 GCATTGACCT ATGGTGTCA AGTCCAGCCC CTCCGCCGGC TGGCCCTTGG CGCGCAGCTT
 5401 GCCCTTGAG GAGGCCCGC ACGAGGGGCA GTGCAGACTT TTGAGGGCGT AGAGCTTGGG
 5461 CGCGAGAAAT ACCGATTCCG GGGAGTAGGC ATCCGCCCG CAGGCCCGC AGACGGTCTC
 5521 GCATTCCACG AGCCAGGTGA GCTCTGGCCG TTGGGGTCA AAAACCAAGGT TTCCCCCATG
 5581 CTTTTGATG CGTTCTTAC CTCTGGTTTC CATGAGCCGG TGTCCACGCT CGGTGACGAA
 5641 AAGGCTGTCC GTGTCCTCGT ATACAGACTT GAGAGGCGT TCCTCGAGCG GTGTTCCCG
 5701 GTCCCTCTCG TATAGAAACT CGGACCACTC TGAGACAAAG GCTCGCGTCC AGGCCAGCAC
 5761 GAAGGAGGCT AAGTGGGAGG GGTAGCGGTG GTTGTCCACT AGGGGGTCCA CTCGCTCCAG
 5821 GGTGTGAAGA CACATGTCGC CCTCTCGGC ATCAAGGAAG GTGATTGGTT TGTAGGTGTA

FIGURE 23
(SHEET 2)

5881 GGCCACGTGA CCGGGTGTTC CTGAAGGGGG GCTATAAAAG GGGGTGGGGG CGCGTTCGTC
 5941 CTCACTCTCT TCCGCATCGC TGTCGCGAG GGCCAGCTGT TGGGGTGAGT ACTCCCCTCG
 6001 AAAAGCGGGC ATGACTTCTG CGCTAAGATT GTCAGTTTCC AAAAACGAGG AGGATTGAT
 6061 ATTCACTCTGG CCCGCCTGTA TGCTTTGAG GGTGCCGCA TCCATCTGGT CAGAAAAGAC
 6121 AATCTTTTIG TTGTCAAGCT TGGTGGCAA CGACCCGTAG AGGGCGTTGG ACAGCAACTT
 6181 GGCGATGGAG CGCAGGGTTT GTTGTGGTC GCGATCGGCG CGCTCCTTGG CGCGATGTT
 6241 TAGCTGCACG TATTCGCGCG CAACGCACCG CCATTGGGA AAGACGGTGG TGCGCTCGTC
 6301 GGGCACCAAGG TGACACGCGC ACCGGGTT GTGCAGGGT ACAAGGTCAA CGCTGGTGGC
 6361 TACCTCTCCG CGTAGGCCT CGTTGGTCCA GCAGAGGCGG CGGCCCTTGC GCGAGCAGAA
 6421 TGGCGGTAGG GGGTCTAGCT GCGTCTCGTC CGGGGGGTCT GCGTCCACGG TAAAGACCCC
 6481 GGGCAGCAGG CGCGCGTCGA AGTAGCTAT CTTCGATCCT TGCAAGTCTA GCGCCTGCTG
 6541 CCATGCGCGG GCGGCAAGCG CGCGCTCGTA TGGGGTGGAGT GGGGGACCCC ATGGCATGGG
 6601 GTGGGTGAGC GCGGAGGCGT ACATGCCCA ATGTCGTAAC CGTAGAGGGG GCTCTCTGAG
 6661 TATTCCAAGA TATGTAGGGT AGCATCTTCC ACCCGGGATG CTGGCGCGCA CGTAATCGTA
 6721 TAGTTCTGTC GAGGGAGCGA GGAGGTCGGG ACCGAGGTTG ATCAGGGCGG GCTGCTCTGC
 6781 TCGGAAGACT ATCTGCCTGA AGATGGCATG TGAGTTGGAT GATATGGTTG GACGCTGGAA
 6841 GACGTTGAAG CTGGCGTCTG TGAGACCTAC CGCGTACCGC ACAGAAGGAGG CGTAGGAGTC
 6901 GCGCAGCTTG TTGACCGACT CGCGGGTGTAC CTGCACGTCT AGGGCGCAGT AGTCCAGGGT
 6961 TTCCCTGATG ATGTCATACT TATCCTGTC CTTCCTTTC CACAGCTCGC GGTTGAGGAC
 7021 AAACCTCTCG CGGTCTTTCC AGTACTCTTG GATCGGAAAC CCGTCGGCCT CGGAACGGTA
 7081 AGAGCCTAGC ATGTAGAACT GGTGACGGC CTGGTAGGCG CAGCATCCCT TTCTACGGG
 7141 TAGCGGTAT GCCTGCGCGG CCTTCGGAG CGAGGTGTGG GTGAGCGCAA AGGTGTCCCT
 7201 GACCATGACT TTGAGGTACT GGTATTTGAA GTCAAGTGTG TGCACTCCGC CCTGCTCCCA
 7261 GAGCAAAAAG TCCGTGCGT TTTTGGAACG CGGATTTGGC AGGGCGAAGG TGACATCGTT
 7321 GAAGAGTATC TTCCCCGCGC GAGGCATAAA GTTGCCTGTG ATGCGGAAGG GTCCCCGGCAC
 7381 CTCGGAACGG TTGTTAATTA CCTGGGGCGC GAGCACGATC TCGTCAAAGC CGTTGATGTT
 7441 GTGGCCCACA ATGTAAAGTT CCAAGAAGCG CGGGATGCC CTTGATGAAAG GCAATTTTTT
 7501 AAGTTCTCG TAGGTGAGCT CTTCAGGGGA GCTGAGCCCG TGCTCTGAAA GGGCCCAGTC
 7561 TGCAAGATGA GGGTTGGAAG CGACGAATGA GCTCCACAGG TCACGGGCCA TTAGCATTTG
 7621 CAGGTGGTCG CGAAAGGTCC TAAACTGGCG ACCTATGGCC ATTTTTCTG GGGTGTGCA
 7681 GTAGAAGGTA AGCGGGTCTT GTTCCCAGCG GTCCCATCCA AGGTTGCGGG CTAGGTCTCG
 7741 CGCGGCAGTC ACTAGAGGCT CATCTCCGCC GAACTTCATG ACCAGCATGA AGGGCACGAG
 7801 CTGCTTCCCA AAGGCCCCCA TCCAAGTATA GGTCTCTACA TCGTAGGTGA CAAAGAGACG
 7861 CTCGGTGCAGA GGATGCGAGC CGATCGGAA GAACTGGATC TCCCGCCACC AATTGGAGGA
 7921 GTGGCTATTG ATGTGGTGAAG AGTAGAAGTC CCTGCGACGG GCGAACACT CGTGCCTGGCT
 7981 TTGTAaaaaa CGTGCAGT ACTGGCAGCG GTGACGGGC TGACGAGGTT
 8041 GACCTGACGA CGCGCACAA GGAAGCAGAG TGGGAATTG AGCCCCCTCGC CTGGGGGGTT
 8101 TGGCTGGTGG TCTCTACTT CGGCTGCTG TCCCTGACCG TCTGGCTGCT CGAGGGGAGT
 8161 TACGGTGGAT CGGACCCACCA CGCCGCGCGA GCCCCAAAGTC CAGATGTCGG CGCGCCGGCG
 8221 TCGGAGCTTG ATGACAACAT CGCGCAGATG GGAGCTGTCC ATGGTCTGGA GCTCCCCCGG
 8281 CGTCAGGTCA GGGGGAGCT CCTGCAGGTT TACCTCGCAT AGACGGGTCA GGGCGCGGGC
 8341 TAGATCCAGG TGATACCTAA TTTCCAGGGG CTGGTTGGTG GGGGGCGTCGA TGGCTTGCAA
 8401 GAGGCCGCAT CCCCCCGGGCG CGACTACGGT ACCCGCGCGC GGGCGGTGGG CGCGGGGGGT
 8461 GTCCCTGGAT GATGCATCTA AAAGCGGTGA CGCGGGCGAG CCCCCGGAGG TAGGGGGGGC
 8521 TCCGGACCCCG CGGGGAGAGG GGGCAGGGG ACGTCGGCGC CGCGCGCGGG CAGGAGCTGG
 8581 TGCTGCGCGC GTAGGTGCT GGCAGACCGG ACGACGCGGC GGTTGATCTC CTGAATCTGG
 8641 CGCCTCTCGC TGAAGACGAC GGGCCCGGTG AGCTTGAGCC TGAAAGAGAG TTCGACAGAA
 8701 TCAATTCTGG TGTGTTGAC GGCAGGGCTGG CGCAAAATCT CTCGACGTC TCCCTGAGTTG
 8761 TCTTGATAGG CGATCTCGGC CATGAACCTGC TCGATCTCTT CCTCTGGAG ATCTCCCGCT
 8821 CCGGCTCGCT CCACGGTGGC GGCAGGGTCC TTGGAAATGC GGGCATGAG CTGCAGAGAAG
 8881 GCGTTGAGGC CTCCCTCGTT CCAGACGCGG CTGTAGACCA CGCCCCCTTC GGCATCGCGG
 8941 GCGCGCATGA CCACCTCGCG GAGATTGAGC TCCACGTGCC GGGCGAAGAC GCGTAGTTT
 9001 CGCAGGGCGCT GAAAGAGGTA GTTGAGGGTG GTGGCGGTGT TTCTGCCAC GAAGAAGTAC
 9061 ATAACCCAGC GTCGAACGT GGATTGTTG ATATCCCCCA AGGCCTCAAG GCGCTCCATG
 9121 GCCTCGTAGA AGTCCACGGC GAAAGTTGAAA AACTGGGAGT TGCGCGCCGA CACGGTTAAC
 9181 TCCCTCTCCA GAAGACGGAT GAGCTCGCG ACAGTGTGCG GCACCTCGCG CTCAAAGGCT
 9241 ACAGGGGCCT TTCTCTCTTC TTCAATCTCC TCTTCCATAA GGGCTCCCC TTCTCTCT

9301 TCTGGCGGCG GTGGGGGAGG GGGGACACGG CGGCACGAC GGCGCACCGG GAGGCGGTGCG
 9361 ACAAAAGCGCT CGATCATCTC CCCCGGGCGA CGGCGCATGG TCTCGGTGAC GGCGCGGCCG
 9421 TTCTCGCGGG GGCGCAGTTG GAAGACGCGG CCCGTCAATGT CCCGGTTATG GGTTGGCGGG
 9481 GGGCTGCCAT CGGGCAGGGG TACGGCGCTA ACGATGCATC TCAACAATTG TTGTGTAGGT
 9541 ACTCCGCCGC CGAGGGACCT GAGCGAGTCC GCATCGACCG GATCGGAAAA CCTCTCGAGA
 9601 AAGGCCTCTA ACCAGTCACA GTCGCAAGGT AGGCTGAGCA CCGTGGCGGG CGGCAGCGGG
 9661 CGGGCGTGG GGTTGTTCT GTGGGAGGTG CTGCTGATGA TGTAATTAAA TAGGCGGTGTC
 9721 TTGAGACGGC GGATGGTCGA CAGAACGACC ATGTCCTTGG GTCCGGCCTG CTGAATGCGC
 9781 AGGCCTGGTGG CCATGCCCA GGCTTCGTTTG TGACATCGC GCAGGTCTTT TAGTAGTGTCT
 9841 TGCGATGAGCC TTTCTACCGG CACTTCTTCT TCTCCCTCCT CTGTCCTGCA ATCTCTTGC
 9901 TCTATCGCTG CGGGCGCGGC GGAGTTTGGC CGTAGGTGGC GCCCTCTTCC TCCCCTCGGT
 9961 GTGACCCCCGAGGCCAGCTCAT CGGCTGAAGC AGGGCTAGGT CGGCAGAAC GCGCTCGGCT
 10021 AATATGGCCT GCTGACCTG CGTGAGGGTA GACTGGAAGT CATCCATGTC CACAAAGCGG
 10081 TGGTATGCGC CGCTGTTGAT GTGTAAAGTG CAGTTGGCCA TAACGGACCA GTAAACGGTC
 10141 TGGTACCCCG GCTGCGAGAG CTGGTGTAC CTGAGACGCG AGTAAGCCCT CGAGTCAAAT
 10201 ACGTAGTCGT TGCAAGTCCG CACCAAGGTG TGGTATCCA CCAAAAGTG CGGGCGCGGC
 10261 TGGCGGTAGA GGGGCCAGCG TAGGGTGGCC GGGGCTCCCG GGGCGAGATC TTCCAACATA
 10321 AGGCATGATGAT ATCCCTAGAT GTACCTGGAC ATCCAGGTGA TGCCGGCGGC GGTGGTGGAG
 10381 GCGCGCGGAA AGTCGCGGAC CGGGTTTCCAG ATGTTGGCGA GCGGCAAAAA GTGCTCCATG
 10441 GTCGGGACGC TCTGGCCGGT CAGGGCGCGC CAATCGTTGA CGCTCTAGCG TGCAAAAGGA
 10501 GAGCTGTAA CGGGCACTC TTCCCTGGTC TGGTGGATAA ATTGCAAGG GTATCATGGC
 10561 GGACGACCGG GTTTCGAGCC CGTATCCGG CGTCCCGCC TGATCCATGC GGTTACCGCC
 10621 CGCGTGTGCA ACCCAGGTGT GCGACGTCAG ACAACGGGG AGTGCTCCTT TTGGCTCCCT
 10681 TCCAGGGCGC CGGGCTGCTG CGCTAGCTTT TTGGCCACT GGGCGCGCGC AGCGTAAGCG
 10741 GTTAAAGCTGG AAAGCGAAAG CATTAAAGTGG CTGCTCCCT GTAGCCGGAG GGTTATTITTC
 10801 CAAGGGTTGA GTCGCGGAC CGGGTTTCG AGTCTCGGAC CGGCCGGACT CGGGCGAACG
 10861 GGGGTTTGGC TCCCCGTAT GCAAGACCCC GCTTCAAAT TCCTCCGAA ACAGGGACGA
 10921 GCCCCCTTTT TGCTTTTCCC AGATGCATCC GGTGCTGCGG CAGATGCGCC CCCCTCCCTA
 10981 GCAGCGGCAAGAGCA GAGCAAGAGC AGCGGCAGAC ATGCAGGGCA CCTCTCCCTC CTCCCTACCGC
 11041 GTCAAGGAGGG CGCACATCCG CGGTTGACGC GGCAGCAGAT GGTGATTACG AACCCCCCGC
 11101 GCGCCGGGCC CGGCACTACC TGGACTTGGG GGAGGGCGAG GGCCCTGGCGC GGCTAGGAGC
 11161 GCCCCCTCTCT GAGGGTACCAAGGGTGA GCTGAAGCGT GATACGCGTG AGGCGTACGT
 11221 GCGCGGGCAG AACCTGTITC GCGACCGCGA GGGAGAGGAG CCCGAGGAGA TCGGGGATCG
 11281 AAAGTTCCAC GCAGGGCGCG AGCTGCGGCA TGGCCTGAAT CGCGAGCGGT TGCTCGCGA
 11341 GGAGGACTTT GAGCCCGACG CGCGAACCGG GATTAGTCCC GCGCGCGCAC ACGTGGCGC
 11401 CGCGGACCTG GTAACCGCAT ACGAGCAGAC GGTGAACCAAG GAGATTAACG TTCAAAAAAG
 11461 CTTAACAAAC CACGTGCGTA CGCTTGTGGC GCGCGAGGAG GTGGCTATAG GACTGATGCA
 11521 TCTGTGGGAC TTTGTAAGCG CGCTGGAGCA AAACCCAAAT AGCAAGCCGC TCATGGCGCA
 11581 GCTGTTCCCT ATAGTGCAGC ACAGCAGGGCAACGAGCA TTCAGGGATG CGCTGCTAAA
 11641 CATAGTAGAG CCCGAGGGCC GCTGGCTGCT CGATTGATA AACATCCCTGC AGAGCATAGT
 11701 GGTGCAGGAG CGCAGCTTGA GCCTGGCTGA CAAGGTGGCC GCCATCAACT ATTCCATGCT
 11761 TAGCCTGGGC AAGTTTACG CCCGCAAGAT ATACCATACC CCTTACGTTT CCATAGACAA
 11821 GGAGGTAAG ATCGAGGGGT TCTACATGCG CATGGCGCTG AAGGTGCTTA CCTTGAGCGA
 11881 CGACCTGGGC GTTTATGCA ACGAGCGCAT CCACAAGGC GTGAGCGTGA GCGGGCGCG
 11941 CGAGCTCAGC GACCGCGAGC TGATGCAAG CCTGCAAAGG GCGCTGGCTG GCACGGCGAG
 12001 CGGGCATAGA GAGGGCGAGT CCTACTTTGA CGGGGGCGCT GACCTGGCGT GGGCCCCAAG
 12061 CGCACCGGCC CTGGAGGCAG CTGGGGCGGG ACCTGGGCTG GCGGTGGCAC CGCGCGCGC
 12121 TGGCAACGTC GGGCGCGTGG AGGAATATGA CGAGGACGAT GAGTACGAGC CAGAGGACGG
 12181 CGAGTACTAA CGGGTGTATGT TTCTGATCAG ATGATGCAAG ACGCAACCGGA CCCGGCGGTG
 12241 CGGGCGGC CGCAGAGCCA GCGCTCCGGC CTTAACCTCA CGGACGACTG GCGCCAGGTC
 12301 ATGGACCGCA TCATGTCGCT GACTGCGGCC AATCCTGACG CGTTCCGGCA GCAGCCGCAG
 12361 GCCAACCGGC TCTCCGCAAT TCTGGAAGCG GTGGTCCCGG CGCGCGCAA CCCCACGCAC
 12421 GAGAAGGTGC TGGCGATCGT AAACCGCGCT GCGCAAACCA GGGCCATCCG GCGCGACGAG
 12481 GCGGGCTGG TCTACGACGC GCTGCTTCAG CGCGTGGCTC GTTACAACAG CGGCAACGTG
 12541 CAGACCAACC TGGACCGGCT GGTGGGGGAT GTGCGCAGG CGTGGCGCA GCGTGAGCGC
 12601 GCGCAGCAGC AGGGCAACCT GGGCTCCATG GTTGCACCAA ACGCCTTCCCT GAGTACACAG
 12661 CCCGCCAACG TGCCCGGGGG ACAGGAGGAC TACACCAACT TTGTGAGCGC ACTGCGGCTA

FIGURE 23
(SHEET 4)

12721 ATGGTGACTG AGACACCGCA AAGTGAGGTG TACCAGTCTG GGCCAGACTA TTTTTTCCAG
 12781 ACCAGTAGAC AAGGCCTGCA GACCGTAAAC CTGAGGCCAGG CTTTCAAAAA CTTGCAAGGGG
 12841 CTGTGGGGGG TGCGGGCTCC CACAGGCAC CGCGCGACCG TGCTCTAGCTT GCTGACGCC
 12901 AACTCGCGCC TGTTGCTGCT GCTAATAGCG CCCTTCACGG ACAGTGGCAG CGTGTCCCG
 12961 GACACATACC TAGGTCACTT GCTGACACTG TACCGCGAGG CCATAGGTCA GGCGCATGTG
 13021 GACGAGCATA CTTTCCAGGA GATTACAAGT GTCAAGCCGCG CGCTGGGGCA GGAGGACACG
 13081 GGCAGCCTGG AGGCAACCCCT AAACCTACCTG CTGACCAACC GGCGGCAGAA GATCCCCTCG
 13141 TTGCACAGTT TAAACAGCGA GGAGGAGCGC ATTTTGCCT ACGTGCAGCA GAGCGTGAGC
 13201 CTTAACCTGA TGCGCGACGG GTAAACGCCG AGCGTGGCGC TGACATGAC CGCGCGCAAC
 13261 ATGGAACCGG GCATGTATGC CTCACAAACGG CCGTTTATCA ACCGCCTAAAT GGACTACTTG
 13321 CATCGCGGG CGCGCGTGA CCCCGAGTAT TTCACCAATG CCATCTTGAA CCCGCACTGG
 13381 CTACCGCCCC CTGGTTCTA CACCGGGGGA TTCAAGGTGC CGCAGGGTAA CGATGGATTG
 13441 CTCTGGGAGC ACATAGACGA CAGCGTGTG TCCCCCGAAC CGCAGACCCCT GCTAGAGTTG
 13501 CAACAGCGCG AGCAGGCGA GGCAGCGCTG CGAAAGGAAA GCTTCCGAG GCCAAGCAGC
 13561 TTGTCCGATC TAGCGCTGC GGCCCCCGGG TCAGATGCTA GTAGCCCAATT TCCAAGCTTG
 13621 ATAGGGTCTC TTACCAAGCAC TCGCACCACC CGCCCGCGC TGCTGGGCGA GGAGGAGTAC
 13681 CTAAACAACT CGCTGCTGCA GCCGCAAGCGC GAAAAAAACC TGCCCTCCGGC ATTTCCAAC
 13741 AACGGGATAG AGAGCCTAGT GGACAAGATG AGTAGATGGA AGACGTACGC GCAGGAGCAC
 13801 AGGGACGTGC CAGGCCCCCGC CCCGCCACC CGTCGTCAA GCCACGACCG TCAGCGGGGT
 13861 CTGGTGTGGG AGGACGATGA CTCGGCAGAC GACAGCAGCG TCCTGGATTG GGGAGGGAGT
 13921 GGCAACCCGT TTGCGCACCT TCGCCCCAGG CTGGGGAGAA TGTTTTAAAA AAAAAAAAGC
 13981 ATGATGCAAA ATAAAAAAACT CACCAAGGGC ATGGCACCGA GCGTTGGTTT TCTTGTATTG
 14041 CCCTTAGTAT GCGGCGCGCG GCGATGTATG AGGAAGGTCC TCCTCCCTCC TACGAGAGTG
 14101 TGGTGAGCGC GGCGCCAGTG GCGGGCGGC TGTTTCTCC CTTCGATGCT CCCCTGGACC
 14161 CGCCGTTGTG GCCTCCGCG TACCTGCGC CTACCGGGGG GAGAAACAGC ATCCGTTACT
 14221 CTGAGTTGGC ACCCCTATTG GACACCACCC GTGTGTACCT GGTGGACAAAC AAGTCAACGG
 14281 ATGTGGCCTC CCTGAACTAC CAGAACGACC ACAGCAACTT TCTGACCAGC GTCATTCAA
 14341 ACAATGACTA CAGCCCCGGG GAGGCAAGCA CACAGACCAT CAATCTTGAC GACCGGTCGC
 14401 ACTGGGGCGG CGACCTGAAA ACCATCTGC ATACCAACAT GCCAAATGTA AACGAGTTCA
 14461 TGTTTACCAA TAAGTTAAG GCGCGGGTGA TGGTGTGCGC CTTCGCTACT AAGGACAATC
 14521 AGGTGGAGCT GAAATACGAG TGGGTGGAGT TCACGCTGCC CGAGGGCAAC TACTCCGAGA
 14581 CCATGACCAT AGACCTTATG AACAAACGCGA TCGTGGAGCA CTACTTGAAA GTGGGCAGAC
 14641 AGAACGGGGT TCTGGAAAGC GACATCGGGG TAAAGTTGTA CACCCGCAAC TTCAGACTGG
 14701 GGTTTGACCC CGTCACTGGT CTTCGTCATGC CTGGGGTATA TACAAACGAA GCCTTCATC
 14761 CAGACATCAT TTTGCTGCCA GGATGCGGGG TGGACTTCAC CCACAGCCGC CTGAGCAACT
 14821 TGTTGGGCAT CCGCAAGCGG CAACCTTCC AGGAGGGCTT TAGGATCACC TACGATGATC
 14881 TGGAGGGTGG TAACATTCCC GCACTGTTGG ATGTGGACGC CTACCAGGCG AGCTTGAAAG
 14941 ATGACACCGA ACAGGGGGGG GGTGGCCGAG GCGGCAGCAA CAGCAGTGGC AGCGGGCGG
 15001 AAGAGAACTC CAACCGGGCA GCGCGGCAA TGCAGCCGGT GGAGGACATG AACGATCATG
 15061 CCATTGCGG CGACACCTTT GCCACACGGG CTGAGGGAGAA GCGCGCTGAG GCCGAAGCAG
 15121 CGGCCGAAGC TGCCGCCCCC GCTGCGCAAC CCGAGGTGCA GAAGCCTCAG AAGAAACCGG
 15181 TGATCAAACCC CTCGACAGAG GACAGCAAGA AACGAGTTA CAACCTAATA AGCAATGACA
 15241 GCACCTTCAC CCAGTACCGC AGCTGGTACCT TTGACATACAA CTACGGCGAC CCTCAGACCG
 15301 GAATCCGCTC ATGGACCCCTG CTTTGCACTC CTGACGTAAC CTGCGGCTCG GAGCAGGTCT
 15361 ACTGGTCGTT GCCAGACATG ATGCAAGACC CCGTGACCTT CCGCTCCACG CGCCAGATCA
 15421 GCAACTTTC CGTGGTGGGC GCGGAGCTGT TGCCCGTGCA CTCCAAGAGC TTCTACAAAG
 15481 ACCAGGCCGT CTACTCCCAA CTCATCCGCC AGTTTACCTC TCTGACCCAC GTGTTCAATC
 15541 GCTTTCCCGA GAACCAGATT TTGGCGCGCC CGCCAGCCCC CACCATCACC ACCGTCAGTG
 15601 AAAACGTTCC TGCTCTCACA GATCACGGGA CGCTACCGCT GCGCAACAGC ATCGGAGGAG
 15661 TCCAGCGAGT GACCATTACT GACGCCAGAC GCGGCACCTG CCCCTACGTT TACAAGGCC
 15721 TGGGCATAGT CTCGCGCGC GTCTTATCGA GCGCAGCTTT TTGAGCAAGC ATGTCATCC
 15781 TTATATCGCC CAGCAATAAC ACAGGCTGGG GCCTGCGCTT CCAAGCAAG ATGTTTGGCG
 15841 GGGCAAGAA GCGCTCCGAC CAACACCCAG TGCGCGTGCG CGGGCAGTAC CGCGCGCC
 15901 GGGCGCGCA CAAACGCGGC CGCACTGGC GCACCAACCGT CGATGACGCC ATCGACGCC
 15961 TGGTGGAGGA GCGCGCAAC TACACGCCA CGCCGCCACC AGTGTCCACA GTGGACGCC
 16021 CCATTCAAGAC CGTGGTGCCTC GGAGCCCGGC GCTATGCTAA AATGAAGAGA CGCGGGAGGC
 16081 CGCTAGCACG TCGCCACCGC CGCCGACCCCG GCACTGCGC CCAACGCGCG GCGGGCGCC

FIGURE 23
(SHEET 5)

16141 TGCTTAACCG CGCACGTCGC ACCGGCCGAC GGGCGGCCAT GGCGGCCGCT CGAAGGCTGG
 16201 CCGGGGTAT TGTCACTGTG CCCCCCAGGT CCAGGCGACG AGCGGCCGCG GCAGCAGCCG
 16261 CGGCCATTAG TGCTATGACT CAGGGTCGCA GGGGCAACGT GTATTGGTG CGCGACTCGG
 16321 TTAGCGGCCT GCGCGTGCCT GTCGCACCC GCCCCCGCG CAACTAGATT GCAAGAAAAA
 16381 ACTACTTAAA CTCGTACTGT TGTATGTATC CAGCGCGGGC GGCGCGCAAC GAAGCTATGT
 16441 CCAAGCGCAA AATCAAAGAA GAGATGCTCC AGGTCACTCGC GCCGGAGATC TATGGCCCCC
 16501 CGAAGAAGGA AGAGCAGGAT TACAAGCCC GAAAGCTAAA GCGGGTCAAA AAGAAAAAGA
 16561 AAGATGATGA TGATGAACCT GACGACGAGG TGGAACTGCT GCACGCTACC GCGCCAGGC
 16621 GACGGGTACA GTGGAAAAGGT CGACGCGTAA AACGTGTTTT GCGAACCGGC ACCACCGTAG
 16681 TCTTTACGCC CGGTGAGCGC TCCACCCGCA CCTACAAGCG CGTGTATGAT GAGGTGTACG
 16741 GCGACGAGGA CCTGCTTGAG CAGGCCAACG AGCGCTCGG GGAGTTGCC TACGGAAAGC
 16801 GGCATAAGGA CATGCTGGCG TTGCGCTGG ACAGAGGGCAA CCCAACACCT AGCCTAAAGC
 16861 CCGTAACACT GCAGCAGGTG CTGCGCGC TTGCACCGTC CGAAGAAAAG CGCGGCCCTAA
 16921 AGCGCGAGTC TGGTGACTTG GCACCCACCG TGCAGCTGAT GGTACCCCAAG CGCCAGCGAC
 16981 TGGAAAGATGT CTTGGAAAAA ATGACCGTGG AACCTGGGCT GGAGGCCGAG GTCCGGCTG
 17041 GGCAATCAA GCAGGTGGCG CCGGGACTGG GCGTGCAGAC CGTGGACGTT CAGATACCCA
 17101 CTACCACTGAC CACCACTATT GCCACCGCCA CAGAGGGCAT GGAGACACAA ACGTCCCCGG
 17161 TTGCGCTACG GGTGGCGGAT GCGCGGTGCG AGGCGGTGCG TCGCGGCCGCG TCCAAGACCT
 17221 CTACGGAGGT GCAAACGGAC CCGTGGATGT TTGCGCTTTC AGCCCCCGG CGCCCGCGCG
 17281 GTTCGAGGAA GTACGGCGCC GCGAGCGC TACTGCCGA ATATGCCCTA CATCCCTCCA
 17341 TTGCGCTTAC CCCCCGGCTAT CGTGGCTACA CCTACCGCCC CAGAAGACGA GCAACTACCC
 17401 GACGCCAAC CACCACTGGA ACCCGCCGCC GCGCTCGCC TCGCCAGCCC GTGCTGGCCC
 17461 CGATTTCCGT GCGCAGGGTG GCTCGCGAAG GAGGCAGGAC CCTGGTGTGCG CCAACAGCGC
 17521 GCTACCACCC CAGCATCGTT TAAAAGCCGG TCTTTGTGGT TCTTGCAGAT ATGGCCCTCA
 17581 CCTGCCGCT CCGTTTCCCG GTGCGGGGAT TCGAGGAAG AATGCACCGT AGGAGGGCA
 17641 TGGCGGGCA CGGCTGACG GCGGGCATGC GTCGTGCAGA CGAACCGGGG CGGCGCGCGT
 17701 CGCACCGTCG CATGCGCGC GGTATCCTGC CCCTCCTTAT TCCACTGATC GCCGCGCGA
 17761 TTGGCGCGT GCCCCGAATT GCATCCGTGG CCTTGCAAGC GOAGAGACAC TGATTTAAA
 17821 CAAGTTGCAT GTGGAAAAAT CAAAATAAAA AGTCTGGACT CTCACGCTCC CTTGGTCTG
 17881 TAACTATTT GTAGAATGGA AGACATCAAC TTGCGTCTC TGGCCCCGGC ACACGGCTCG
 17941 CGCCCGTCA TGGGAAACTG GCAAGATATC GGCACCAGCA ATATGAGCGG TGGGCCCTTC
 18001 AGCTGGGCT CGCTGTGGAG CGGCATTAAA AATTTGGTCCACCGTTAA GAACTATGGC
 18061 AGCAAGGCTT GGAACAGCAG CACAGGCCAG ATGCTGAGGG ATAAGTTGAA AGAGCAAAT
 18121 TTCCAACAAA AGGTGGTAGA TGGCTGGCC TCTGGCATTAA GCGGGGTGGT GGACCTGGCC
 18181 AACCAGGCA TGAAAATAA GATTAACAGT AAGCTTGATC CCCGCCCTCC CGTAGAGGAG
 18241 CCTCCACCGG CCGTGGAGAC AGTGTCTCCA GAGGGCGTG GCGAAAAGCG TCCGCCCGCC
 18301 GACAGGGAAAG AACTCTGGT GACGCAAATA GACGAGCCTC CCTCGTACGA GGAGGCACTA
 18361 AAGCAAGGCC TGCCACCAAC CCGTCCCAC GCGCCCATGG CTACCGGAGT GCTGGGCCAG
 18421 CACACACCG TAACGCTGGA CCTGCTCCC CCCGCCGACA CCCAGCAGAA ACCTGTGCTG
 18481 CCAGGCCCGA CCGCGTTGT TGTAACCGT CCTAGGCCGCG CGTCCCTGCG CGCGCCCGCC
 18541 AGCGGTCCGC GATCGTTGCG GCCCGTAGCC AGTGGCAACT GCGAAAGCAC ACTGAACAGC
 18601 ATCGTGGTC TGGGGGTGCA ATCCCTGAAG CGCCGACGAT GCTTCTGAAT AGCTAACGTG
 18661 TCGTATGTGT GTCACTGTATG CGTCCATGTC GCGCGCAGAG GAGCTGCTGA GCCGCCGCGC
 18721 GCCCCCTTTC CAAGATGGCT ACCCCCTTCA TGATGGCGCA GTGGTCTTAC ATGCACATCT
 18781 CGGGCCAGGA CGCCTCGGAG TACCTGAGCC CGGGGTGGT GCGAGTTGCC CGCGCCACCG
 18841 AGACGTACTT CAGCGTGAAT AACAGTTAA GAAACCCAC CGTGGGCCCT ACGCACGAG
 18901 TGACCACAGA CGGGTCCCAG CGTTTGACGC TGCGGTTCAT CCTGTGGAC CGTGAGGATA
 18961 CTGCGTACTC GTACAAGGGCG CGGTTCACCC TAGCTGTGGG TGATAACCGT GTGCTGGACA
 19021 TGGCTTCCAC GTACTTTGAC ATCCCGGGCG TGCTGGACAG GGGGCTACT TTTAAGCCCT
 19081 ACTCTGGCAC TGCCCTACAAAC GCCCTGGCTC CCAAGGGTGC CCCAAATCCT TGCGAATGGG
 19141 ATGAAGCTGC TACTGCTCTT GAAATAAACC TAGAAGAAGA GGACGATGAC AACGAAGACG
 19201 AAGTAGACGA GCAAGCTGAG CAGCAAAAAA CTCACGTATT TGCGCAGGCG CCTTATTCTG
 19261 GTATAAAATAT TACAAAGGGAG GGTATTCAA TAGGTGTCGA AGGTCAAACCA CCTAAATATG
 19321 CCGATAAAAC ATTCAACCT GAACTCTAAA TAGGAGAATC TCAGTGGTAC GAAACTGAAA
 19381 TTAATCATGC AGCTGGGAGA GTCCCTAAAAA AGACTACCCC AATGAAACCA TGTTACGGTT
 19441 CATATGCAAA ACCCACAAAT GAAAATGGAG GGCAAGGCAT TCTTGTAAAG CAACAAAATG
 19501 GAAAGCTAGA AAGTCAGTG GAAATGCAAT TTTCTCAAC TACTGAGGCG ACCGCAGGCA

19561 ATGGTGATAA CTTGACTCCT AAAGTGGTAT TGTACAGTGA AGATGTAGAT ATAGAAACCC
 19621 CAGACACTCA TATTCTTAC ATGCCCACTA TTAAGGAAGG TAACTCACGA GAACTAATGG
 19681 GCCAACAAAC TATGCCCAAC AGGCCTAATT ACATTGCTT TAGGGACAAT TTTATTGGTC
 19741 TAATGTATTA CAACAGCACG GGTAATATGG GTGTTCTGGC GGGCAAGCA TCGCAGTGA
 19801 ATGCTGTTGT AGATTGCAA GACAGAAACA CAGAGCTTC ATACCAGCTT TTGCTTGATT
 19861 CCATTGGTGA TAGAACCAAGG TACTTTCTA TGTGGAATCA GGCTGTTGAC AGCTATGATC
 19921 CAGATGTTAG AATTATTGAA AATCATGGAA CTGAAGATGA ACTTCCAAT TACTGCTTTC
 19981 CACTGGGAGG TGTGATTAAT ACAGAGACTC TTACCAAGGT AAAACCTAAA ACAGGTCAGG
 20041 AAAATGGATG GGAAAAAGAT GCTACAGAAAT TTTCAGATAA AAATGAAATA AGAGITGGAA
 20101 ATAATTTCG CATGGAATC AATCTAAATG CCAACCTGTG GAGAAATTTC CTGTAACCCA
 20161 ACATAGCGCT GTATTGCCC GACAAGCTAA AGTACAGTCC TTCCAACGTA AAAATTCTG
 20221 ATAACCCAAA CACCTACGAC TACATGAACA AGCGAGTGGT GGCTCCCGGG TTAGTGGACT
 20281 GCTACATTAA CCTTGAGCA CGCTGGTCCC TTGACTATAT GGACAACGTC AACCCATTAA
 20341 ACCACCACCG CAATGCTGGC CTGCGCTACC GCTCAATGTT GCTGGCAAT GGTGCTATG
 20401 TGCCCTTCCA CATCCAGGTG CCTCAGAAGT TCTTGGCCAT TAAAAACCTC CTTCTCTGC
 20461 CGGGCTCATC CACCTACGAG TGGAACTTCA GGAAGGATGT TAACATGGTT CTGCAGAGCT
 20521 CCCTAGGAAA TGACCTAAGG GTTGACAGGAG CGAGCATTAA GTTGTATAGC ATTTGCTT
 20581 ACGCCACCTT CTTCCTCATG GCCCACAACA CGGCCTCCAC GCTTGAGGCC ATGCTTAGAA
 20641 ACGACACCAA CGACCAAGTCC TTTAACGACT ATCTCTCCG CGCCAACATG CTCTACCTA
 20701 TACCCGCCAA CGCTACCAAC GTGCCCCATAT CCATCCCCCTC CCGCAACTGG GCGGCTTCC
 20761 GCGGCTGGC CTTCACGGC CTTAAGACTA AGGAAACCCC ATCACTGGC TCGGGCTACG
 20821 ACCCTTATTA CACCTACTCT GGCTCTATAC CCTACCTAGA TGGAACCTTT TACCTCAACC
 20881 ACACCTTAA GAAGGTGGCC ATTACCTTIG ACTCTCTGT CAGCTGGCCT GGCAATGACC
 20941 GCCTGCTTAC CCCAACGAG TTGAAATTAA AGCGCTCAGT TGACGGGGAG GGTTACAACG
 21001 TTGCCCAGTG TAACATGACC AAAGACTGGT TCCGTGACA AATGCTAGCT AACTACAACA
 21061 TTGGCTACCA GGGCTTCTAT ATCCCAGAGA GCTACAAGGA CGCGATGTAC TCCCTCTTA
 21121 GAAACCTCCA GCCCATGAGC CGTCAGGTGG TGGATGATAC TAAATACAAG GACTACCAAC
 21181 AGGTGGGCAT CCTACACCAA CACAACAACG CTGGATTGT TGGCTACCTT GCCCCCACCA
 21241 TGCGCGAAGG ACAGGCCTAC CCGCTAACT TCCCCTATCC GCTTATAGGC AAGACCGCAG
 21301 TTGACAGCAT TACCCAGAAA AGTTTCTTT GCGATCGCAC CCTTGGCGC ATCCCATTCT
 21361 CCAGTAACCT TATGCCATG GGCGCACTCA CAGACCTGGG CCAAAACCTT CTCTACGCCA
 21421 ACTCCGCCA CGCGCTAGAC ATGACTTTTG AGGTGGATCC CATGGACGAG CCCACCCCTC
 21481 TTATGTTT GTTGAGTC TTGACGTGG TCCGTGCA CCGGCGCAG CGCGCGTCA
 21541 TCGAAACCGT GTACCTGCGC ACGCCCTCT CGGCGGCAA CGCCACAACA TAAAGAAGCA
 21601 AGCAACATCA ACAACAGCTG CCGCCATGGG CTCCAGTGG CAGGAACGTGA AAGCCATTGT
 21661 CAAAGATCTT GGTGTTGGGC CATATTTTTT GGGCACCTAT GACAAGCGCT TTCCAGGCTT
 21721 TGTTCTCCA CACAAGCTCG CCTGCGCCAT AGTCAATAGC GCCGGTGCAG AGACTGGGG
 21781 CGTACACTGG ATGGCCCTTG CCTGGAACCC GCACTAAAA ACATGCTACC TCTTGGAGCC
 21841 CTTGGCTTT TCTGACCAGC GACTCAAGCA GGTGTTACAG TTGAGTACG AGTCACTCCT
 21901 GCGCCGTAGC GCCATTGCTT CTTCCCCCGA CGCTGTATA ACGCTGGAAA AGTCCACCCA
 21961 AAGCGTACAG GGGCCAACG CGGCCGCGCTG TGGACTATTG TGCTGCATGT TTCTCCACGC
 22021 CTTTGCAAC TGGCCCCAAA CTCCCATGGA TCACAACCCC ACCATGAACC TTATTACCGG
 22081 GGTACCCAAAC TCCATGCTCA ACAGTCCCCA GGTACAGCCC ACCCTGCGTC GCAACCCAGGA
 22141 ACAGCTCTAC AGCTTCTGG AGCGCCACTC GCCCTACTTC CGCAGGCCACA GTGCGCAGAT
 22201 TAGGAGCGCC ACTTCTTTT GTCACTTGAA AAACATGTA AAAATATGTA CTAGAGACAC
 22261 TTCAATAAA GGCAAATGCT TTATTTGTA CACTCTCGGG TGATTATTTA CCCCCACCC
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 22441 CGGCAGCTCG GTGAAGTTT CACTCCACAG GCTGCGCACC ATCACCAACG CGTTAGCAG
 22501 GTCGGGCGCC GATATCTTGA AGTCCAGTT GGGGCGCTCCG CCCCTGCGCGC GCGAGTTGCG
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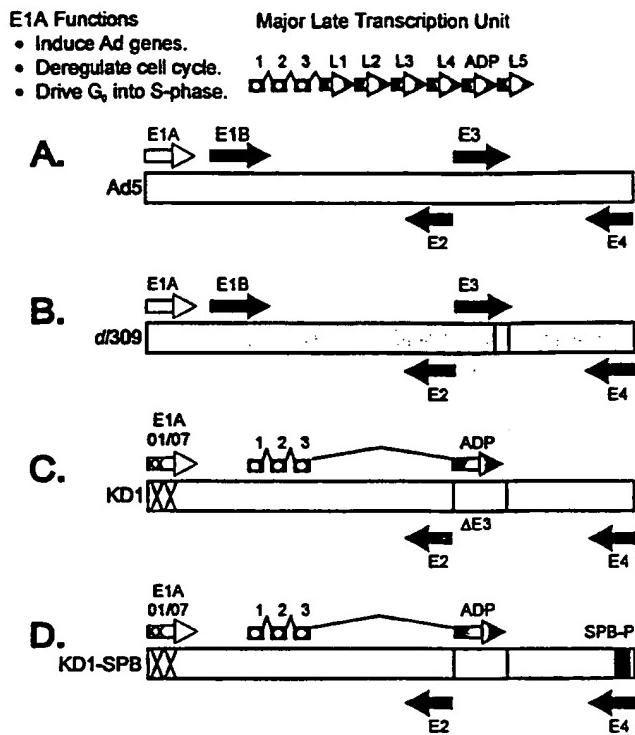
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 23161 AAACGACTGC AGGTACGCCG GCAGGAATCG CCCCCATCATC GTCACAAAAGG TCTTGTGCT
 23221 GGTGAAGGTC AGCTGCAACC CGCGGTGCTC CTCGTTCAAGC CAGGTCTTGC ATACGGCCGC
 23281 CAGAGCTTCC ACTTGGTCAG GCAGTAGTTT GAAGTTCGCC TTAGATCGT TATCCACGTG
 23341 GTACTTGTCC ATCAGCGCAG GCGCAGCTC CATGCCCTTC TCCCACCGAG ACACGATCGG
 23401 CACACTCAGC GGGTTCATCA CGTAAATTTC ATTTCCGCT TCGCTGGGCT CTTCCTCTTC
 23461 CTCTTGCCTC CGCATACAC GCGCCACTGG GTCTGCTTCA TTCAAGCCGC GCACTGTGCG
 23521 CTTACCTCTT TTGCCATGCT TGATTAGCAC CGGGGGGGTG CTGAAACCCA CCATTGTAG
 23581 CGCCACATCT TCTCTTCTT CTCGCTGTC CACGATTACC TCTGGTGTAG GCGGGCGCTC
 23641 GGGCTTGGGA GAAGGGCGCT TCTTTTCTT CTTGGGCGCA ATGGCCAAAT CCGCCGCCGA
 23701 GGTGATGGC CGCGGGCTGG GTGTGCGCG CACCAAGCGC TCTTGTGATG AGTCTTCCTC
 23761 GTCTCGGAC TCGATACGCC GCCTCATCCG CTTTTTGGGG GGCGCCCGGG GAGGCGGGCG
 23821 CGACGGGGAC GGGGACGACA CGTCCTCCAT GGTTGGGGGA CGTCGCGCCG CACCGCGTCC
 23881 GCGCTCGGGG GTGGTTTCGC GCTGCTCTC TTCCCGACTG GCCATTCTC TCTCCTATAG
 23941 GCAGAAAAAG ATCATGGAGT CAGTCGAGAA GAAGGACAGC CTAACCGCCC CCTCTGAGTT
 24001 CGCCACCACCG GCCTCCACCG ATGCCGCAA CGCGCCTTAC ACCTTCCCCG TCGAGGCACC
 24061 CCCGTTGAG GAGGAGGAAG TGATTATCGA GCAGGACCCA GGTTTGTAA GCGAAGACGA
 24121 CGAGGACCGC TCAGTACCAA CAGAGGATAA AAAGCAAGAC CAGGACAACG CAGAGGCAAA
 24181 CGAGGAACAA GTCGGGCGGG GGGACGAAAG GCATGGCGAC TACCTAGATG TGGGAGACGA
 24241 CGTGTGTTG AAGCATCTGC AGCGCCAGTG CGCCATTATC TGCGACGCGT TGCAAGAGCG
 24301 CAGCGATGTG CCCCTCGCCA TAGCGGATGT CAGCCTTGCC TACGAACGCC ACCTATTCTC
 24361 ACCGCGCGTA CCCCCAACAC GCCAAGAAAA CGGCACATGC GAGCCCAACC CGCGCTCAA
 24421 CTTCTACCCC GTATTTGCCG TGCCAGAGGT GCTTGCACC TATCACATCT TTTTCAAAA
 24481 CTGCAAGATA CCCCTATCCT GCGGTGCAA CCGCAGCCGA CGGGACAAGC AGCTGGCCTT
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 24721 AAAACGCAGC ATCGAGGTCA CCCACTTGC CTACCCGGCA CTTAACCTAC CCCCCAAGGT
 24781 CATGAGCACA GTCATGAGTG AGCTGATCGT GCGCGTGC CAGCCCCCTGG AGAGGGATGC
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 24961 AGTGCTCGTT ACCGGTGGAGC TTGAGTGAT GCAGCGGTTT TTGCTGACCG CGGAGATGCA
 25021 GCGCAAGCTA GAGGAAACAT TGCACATAC CTTTCGACAG GGCTACGTAC GCCAGGCTG
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 25681 CTCCCTGGTT TGCAATTGCG AGCTGTTAA CGAAAGTCAA ATTATCGGTA CCTTGTAGCT
 25741 GCAGGGTCCC TCGCTGACG AAAAGTCCGC GGCTCCGGGG TTGAAACTCA CTCCGGGGCT
 25801 GTGGACGTG GCTTACCTTC GCAAATTGT ACCTGAGGAC TACCAACGCC ACGAGATTAG
 25861 GTTCTACGAA GACCAATCCC GCGCGCCAAA TGCGGAGCTT ACCGGCTGCG TCATTACCA
 25921 GGGCCACATT CTGGCCAAT TGCAAGCCAT CAACAAAGCC CGCGAAGAGT TTCTGCTAG
 25981 AAAGGGACGG GGGTTTACT TGGACCCCA GTCCGGCGAG GAGCTCAACC CAATCCCCC
 26041 GCCGCCGCAG CCCTATCAGC AGCAGCCCG GGCCTTGCT TCCCAGGATG GCACCCAAA
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 26221 AAGCTCCGA GGTGCAAGAG GTGTGAGC AAACACCGTC ACCCTCGGTG GCATTCCCCCT
 26281 CGCCGGCGCC CCAGAAATCG GCAACCGTT CCAGCATGGC TACAACCTCC GCTCCTCAGG
 26341 CGCCGCGCGC ACTGCCGTT CGCGGACCCA ACCGTAGATG GGACACCAACT GGAACCGAGG

26401 CCGGTAAGTC CAAGCAGCCG CCGCCGTAG CCCAAGAGCA ACAACAGCGC CAAGGCTACC
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 26581 TGCATTACTA CCGTCATCTC TACAGCCCAT ACTGCACCGG CGGCAGCGGC AGCGGCAGCA
 26641 ACAGCAGCGG CCACACAGAA GCAAAGGCAGA CCGGATAGCA AGACTCTGAC AAAGCCAAG
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 26761 GTATCGACCC GCGAGCTTAG AAACAGGATT TTTCCTACTC TGATGCTAT ATTTCAACAG
 26821 AGCAGGGGCC AAGAACAAAG GCTGAAAATA AAAAACAGGT CTCTGCGATC CCTCACCCGC
 26881 AGCTGCCCTGT ATCACAAAAAG CGAAGATCAG CTTCGGCGCA CGCTGGAAGA CGCGGAGGCT
 26941 CTCTTCAGTA AATACTGCGC GCTGACTCTT AAGGACTAGT TTGCGCCCTT TTCTCAAATT
 27001 TAAGCGCGA AACTACGTCA TCTCCAGCGG CCACACCCGG CGCCAGCACC TGTCGTCAGC
 27061 GCCATTATGA GCAAGGAAAT TCCCACGCCG TACATGTGGA GTTACCCAGGC ACAAAATGGGA
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 27241 CGGGCTATTAA CCACACACCC TCGTAATAAC CTTAATCCCC GTAGTTGGCC CGCTGCCCTG
 27301 GTGTACCAAGG AAAGTCCCGC TCCCACCACT GTGGTACTTC CCAGAGACGC CCAGGCCAA
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 29641 AAATGGGCAA CGGGCTCTCT CTGGACGAGG CGGGCAACCT TACCTCCCAA AATGTAACCA
 29701 CTGTGAGGCC ACCTCTCAAA AAAACCAAGT CAAACATAAA CCTGGAAATA TCTGCACCC
 29761 TCACAGTTAC CTCAGAAGCC CTAACGTGG CTGCGCCCGC ACCTCTAATG GTCGCGGGCA

29821 ACACACTCAC CATGCAATCA CAGGCCCGC TAACCGTGC CGACTCCAAA CTTAGCATTG
 29881 CCACCCAAGG ACCCCCTACA GTGTAGAAG GAAAGCTAGC CCTGCAAACA TCAGGGCCCC
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**FIGURE 24**

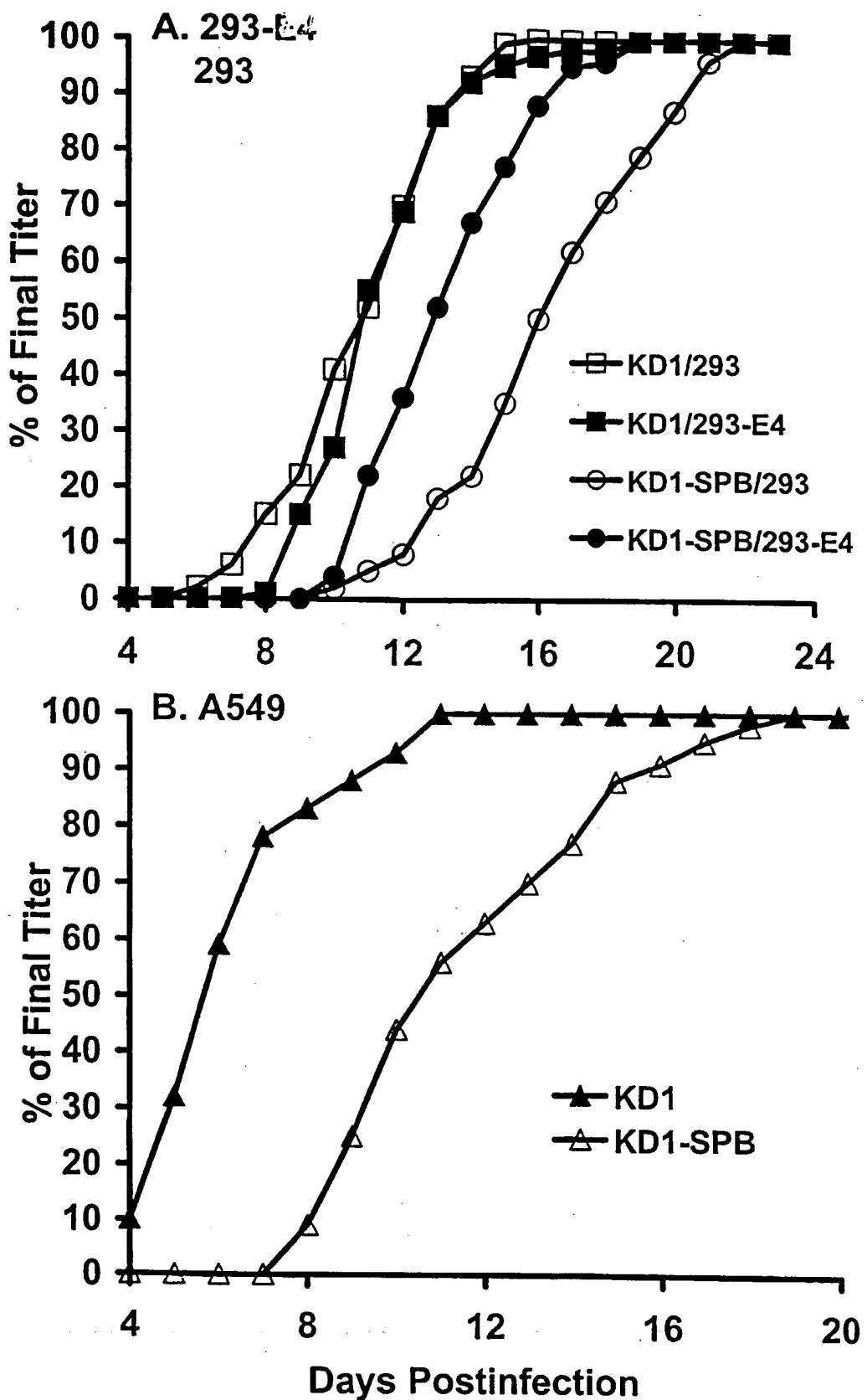


FIGURE 25

S7 | 66

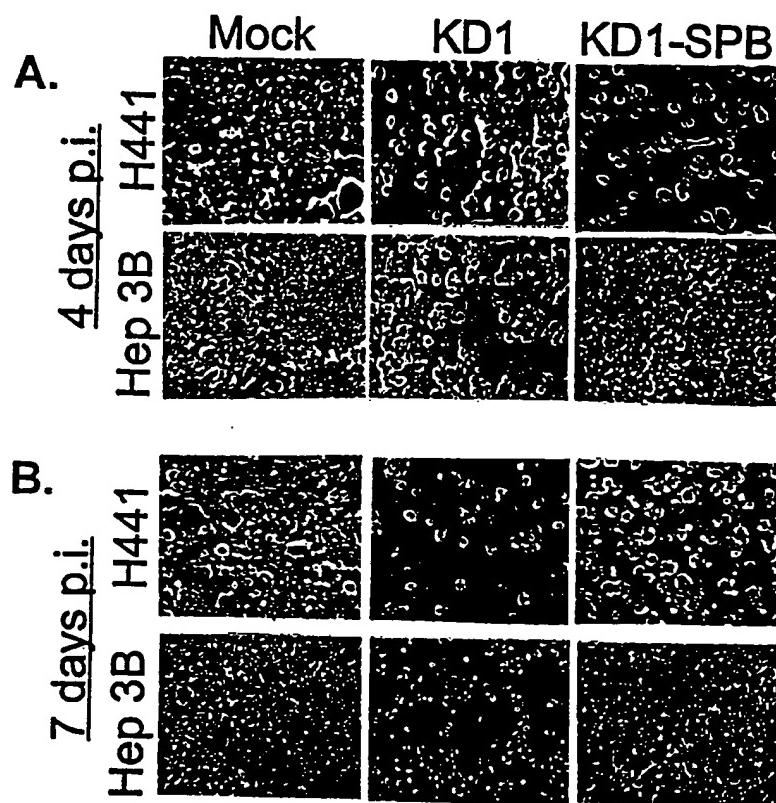


FIGURE 26

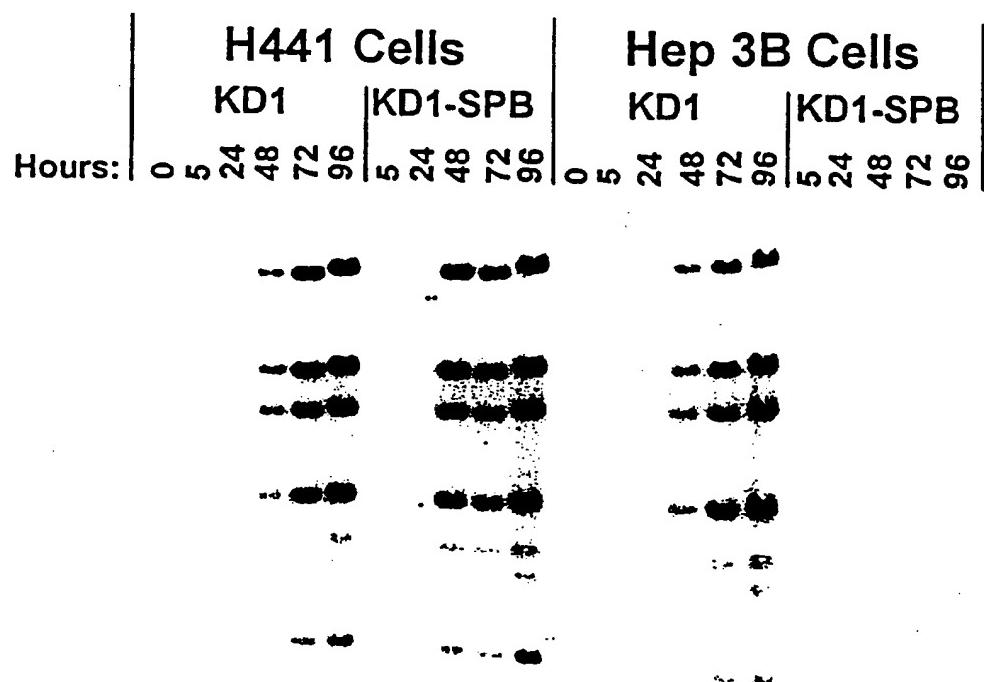


FIGURE 27A

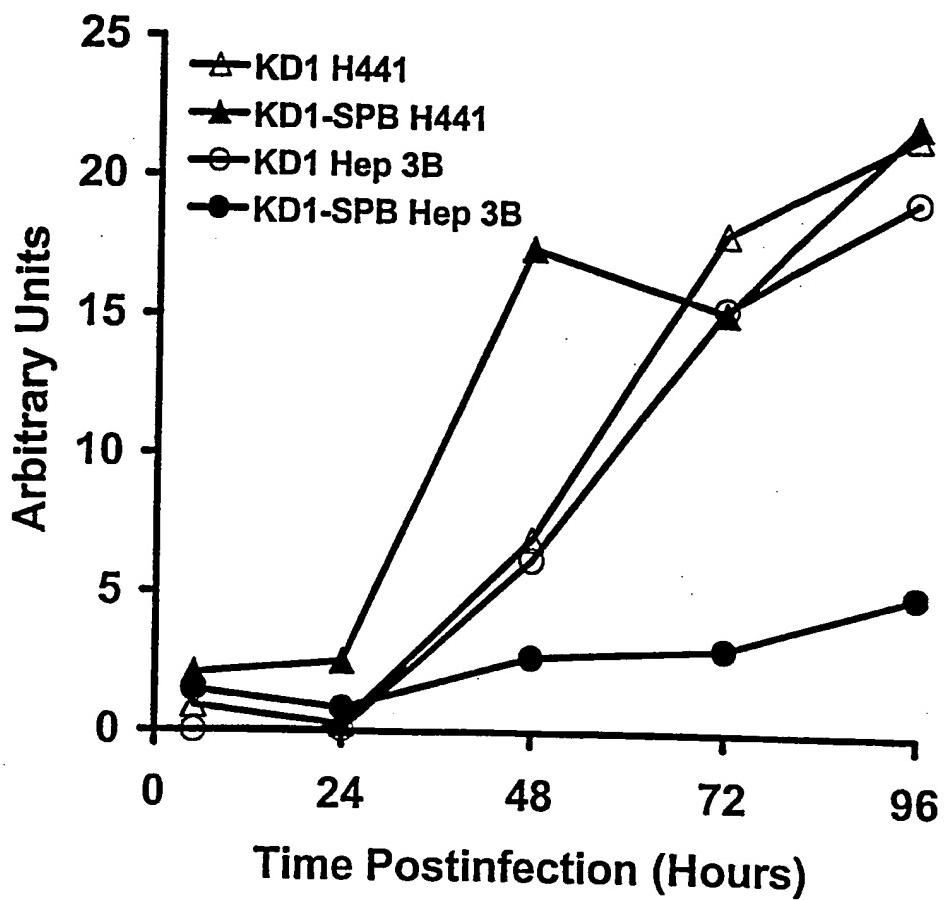


FIGURE 27B

60 | 66

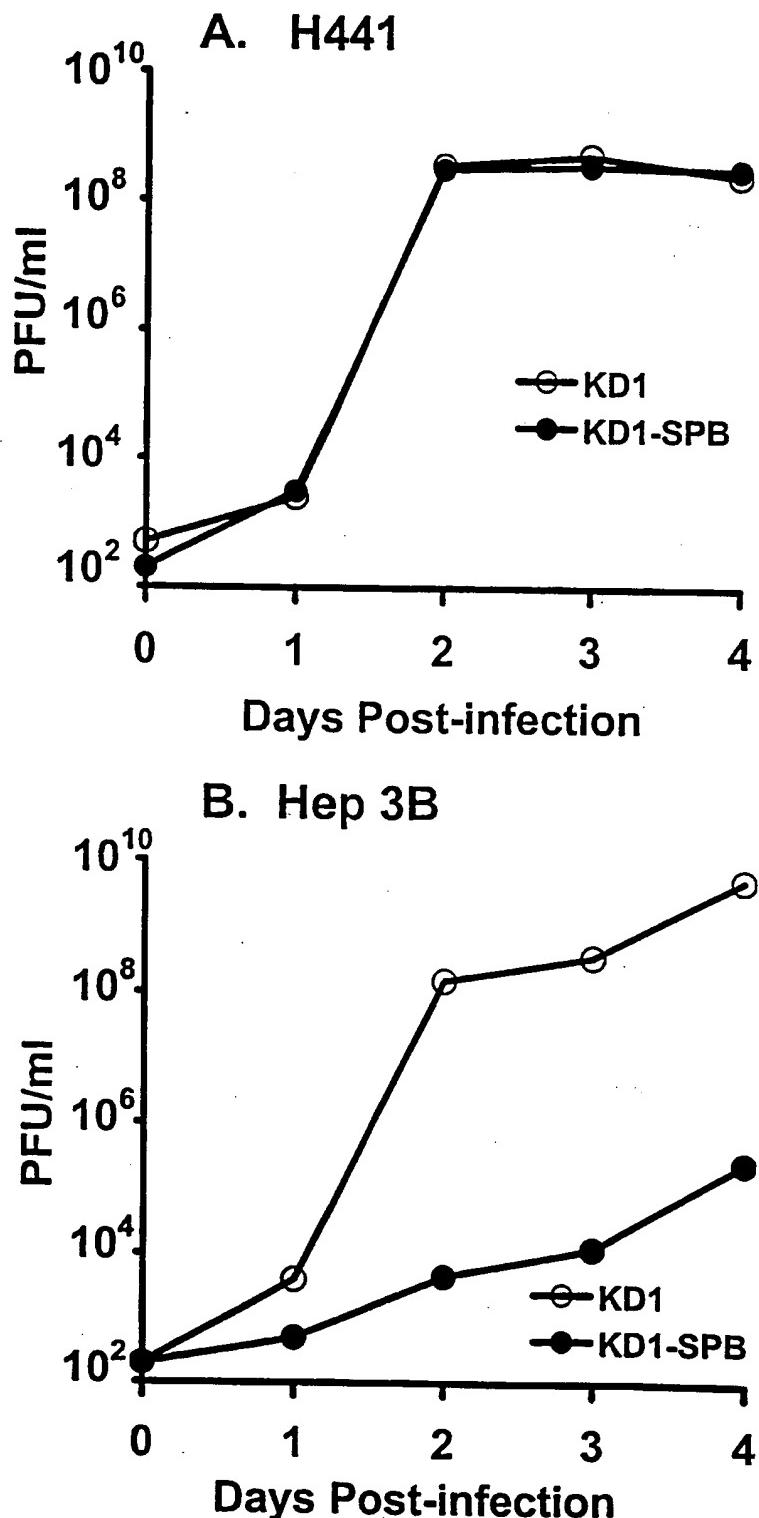


FIGURE 28

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FIGURE 29

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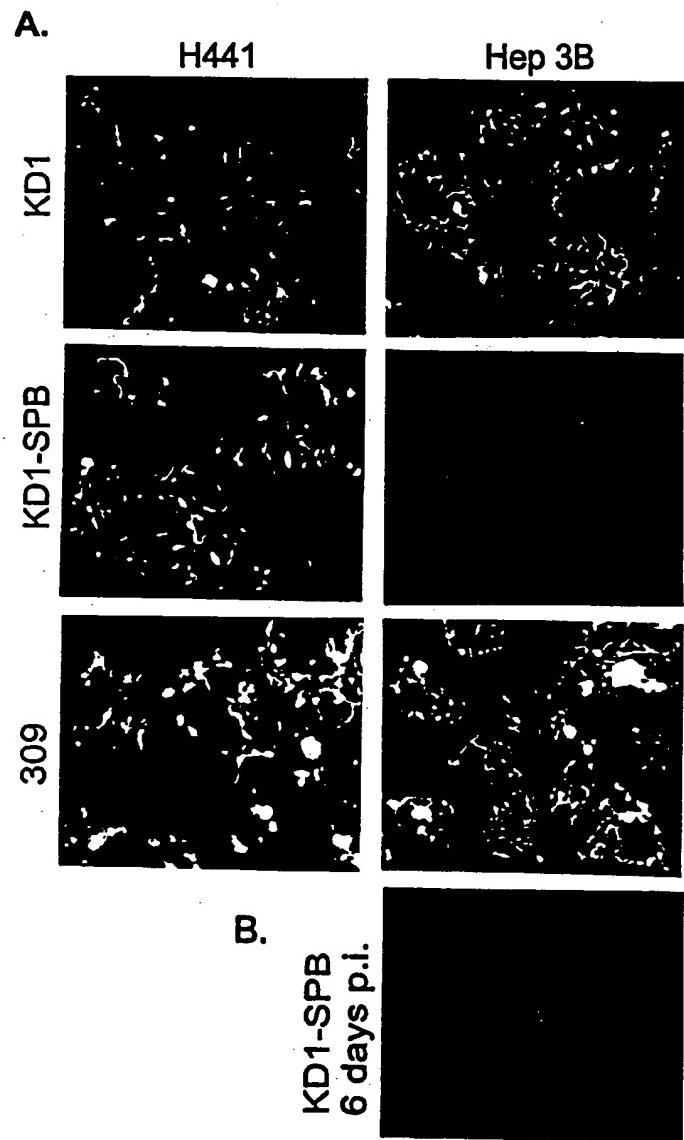


FIGURE 30

63 | 66

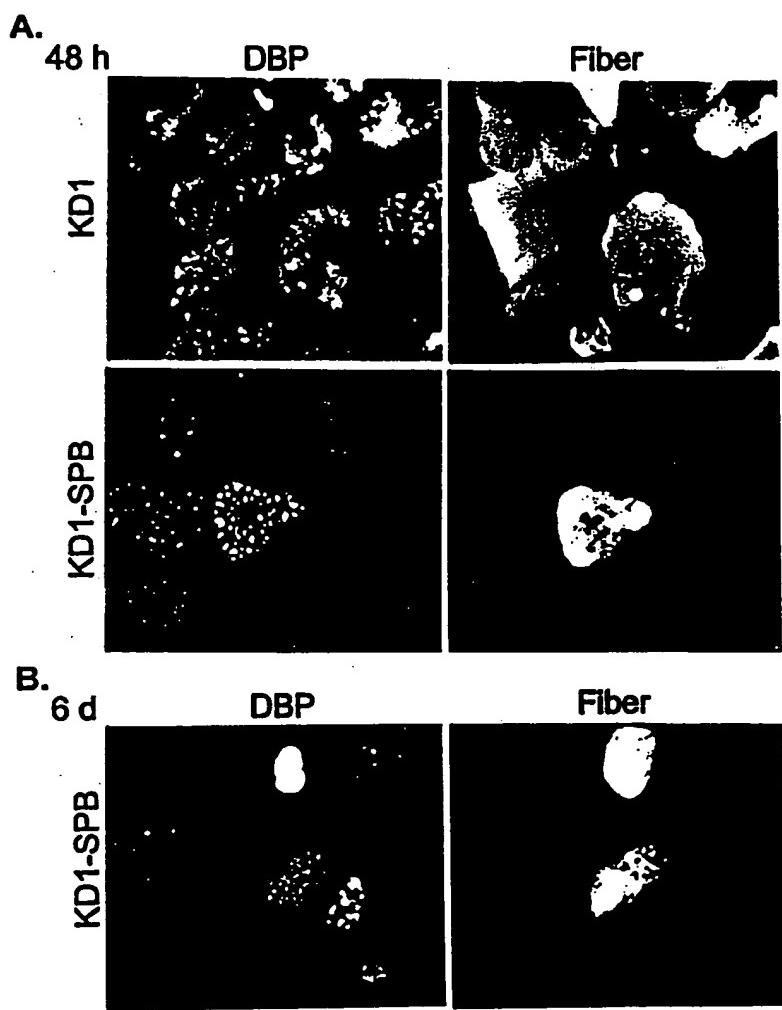
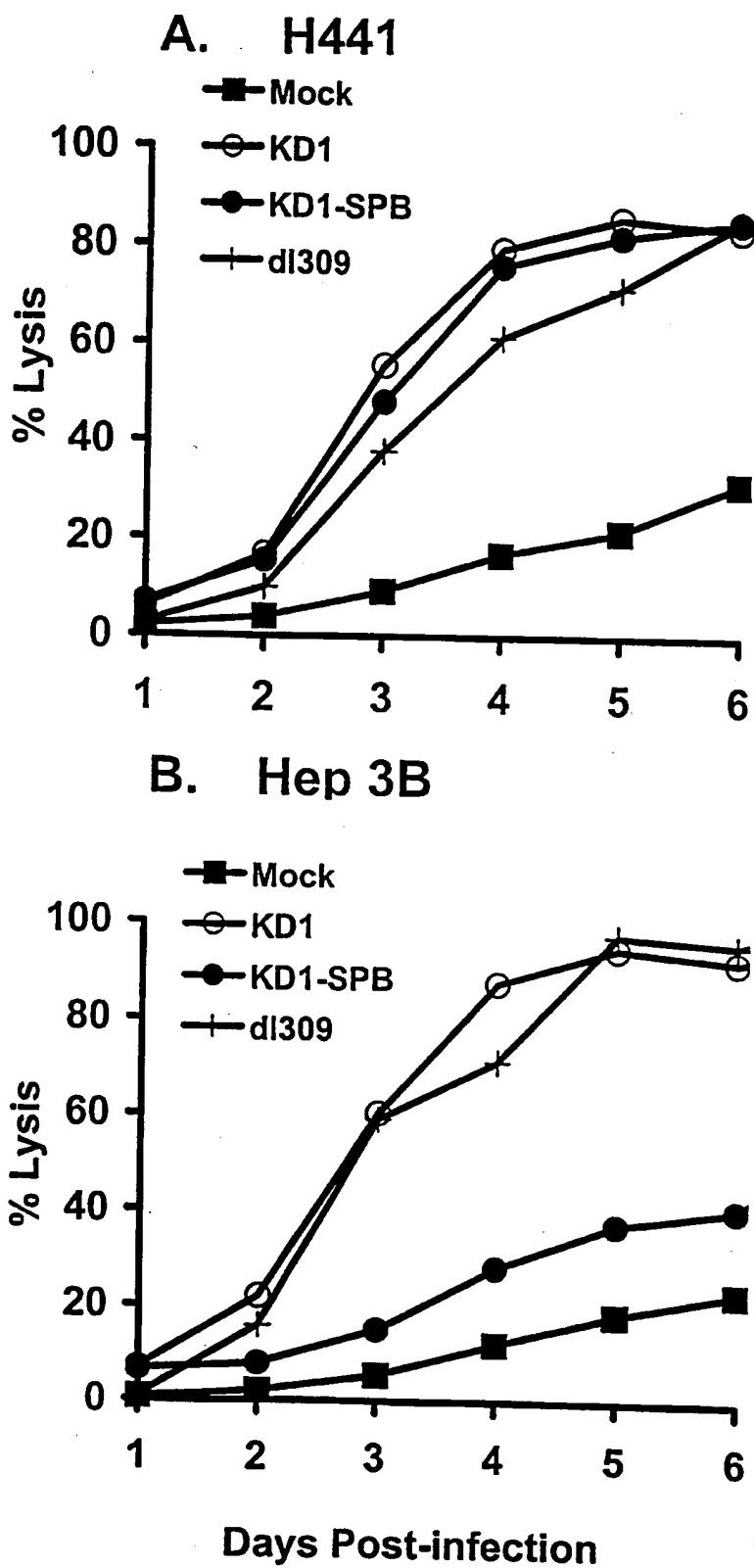
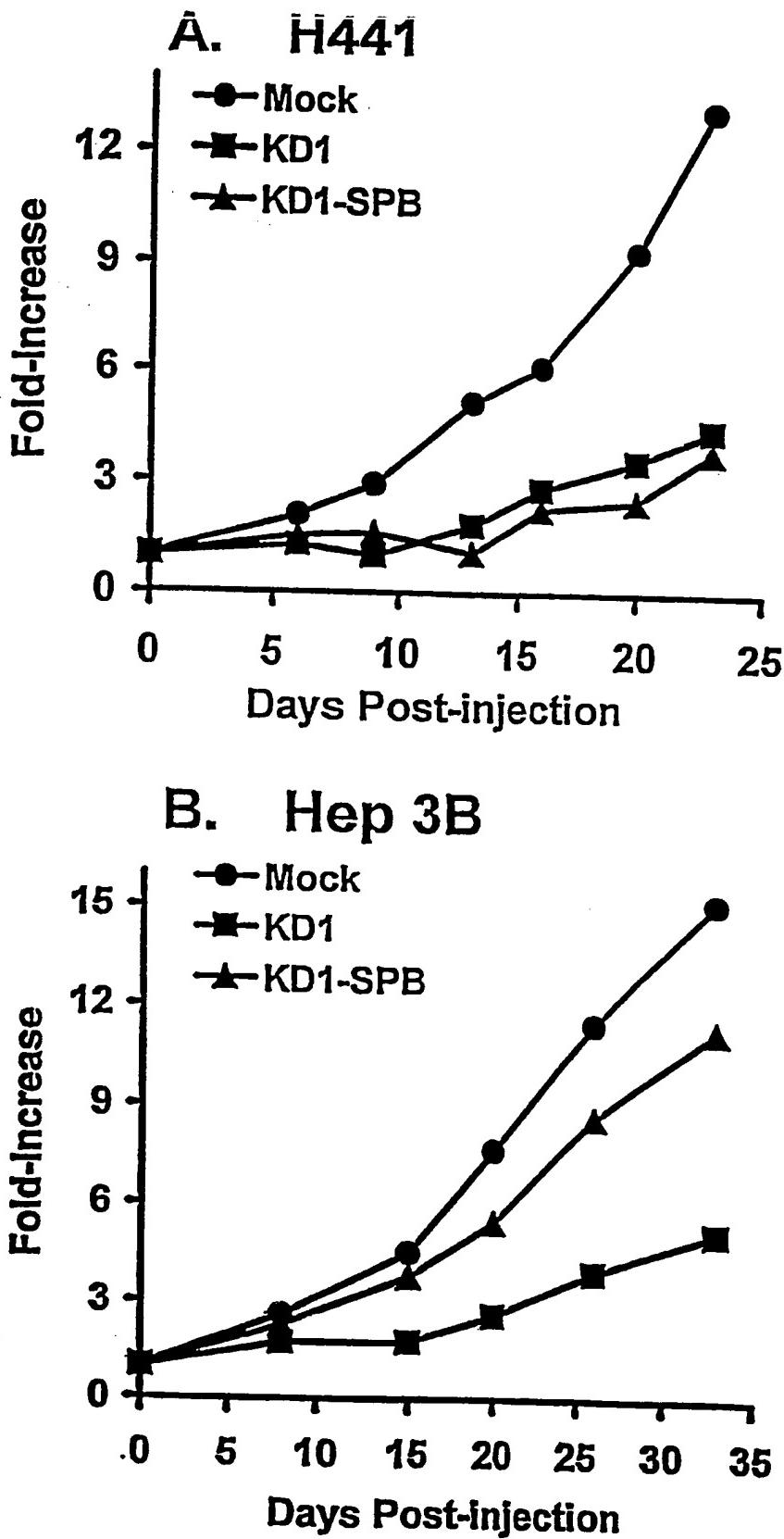


FIGURE 31

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**FIGURE 32**

65166

**FIGURE 33**

66/66

SEQUENCE LISTING

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Toth, Karoly
Doronin, Konstantin
Tollefson, Ann E.

<120> Replication-Competent Anti-Cancer Vectors

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